# SYSTEM

OF

# DISEASES OF THE EYE.

BY

AMERICAN, BRITISH, DUTCH, FRENCH, GERMAN, AND SPANISH AUTHORS.

EDITED BY

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# SYSTEM

OF

# DISEASES OF THE EYE.

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## PART IV.

ANOMALIES OF MOTOR APPARATUS, DISEASES OF CORNEA AND LENS, AMETROPIA, MEDICAL OPHTHALMOLOGY.

# ANOMALIES OF THE MOTOR APPARATUS OF THE EYES.

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## I.—PARALYSIS OF THE OCULAR MUSCLES.

In the study of this subject, we must keep in mind the origin and insertion of the muscle, as well as its plane of action, which passes through these two points and the centre of rotation of the eye.[1] If we recall the fact that the muscle by its contraction brings its points of insertion and origin towards each other, we shall know not only the influence which the muscle exercises on the eyeball under normal conditions, but also the symptoms which result from its paralysis.

*General Symptomatology.*—The first consequence of paralysis of a muscle of the eye is a diminution of the rotation of the globe in the direction in which the muscle normally causes the eyeball to turn.

The limitation of the field of fixation,[2] thus produced, is more pronounced in proportion to the completeness of the paralysis, and is more characteristic in proportion as the muscle in question is the only one to act in a certain direction. Thus the rotations around the vertical axis towards the temple and the nose are accomplished almost exclusively by the external and internal recti muscles. Hence paralysis of the external rectus muscle almost entirely annihilates the abduction of the eye and produces a considerable gap in the temporal side of the field of fixation.

The same is not absolutely the case for the superior and inferior recti

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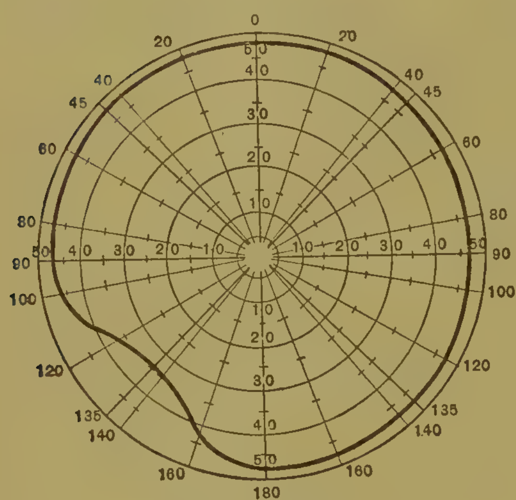
[1] In the case of the superior oblique, the pulley through which it passes must be considered as its origin.

[2] By "field of fixation" we understand the space which an eye dominates by its movements, or the aggregation of all the points in space towards which the line of sight of an eye may be directed while the head remains motionless. The limits of the fixation-field correspond, therefore, to the limits of the excursions of the eye. They extend, in all directions, about forty-seven degrees. See Fig. 1, which represents the minimum field of fixation of a right eye. Here the field is limited much within and below by the nose.

and oblique muscles, because their action is almost always a combined one. For rotation about the transverse axis, for instance,—the raising and lowering of the eye,—at least two muscles act together. Looking downward is accomplished by the combined action of the inferior rectus and the superior oblique muscle; looking upward, by that of the superior rectus and the inferior oblique.

Fig. 1.




Normal field of fixation of a right eye. (Landolt.)

If there be paresis or even paralysis of one of the depressors,—of the superior oblique, for instance,—a lessening in extent of the lower part of the field of fixation will be discoverable; but this lack of action will never be so great as that on the temporal side produced by paralysis of the abductor, because the inferior rectus muscle has also to do with looking downward. It is true that, by its own action, the superior oblique muscle causes the eye to turn not only downward, but also outward. Hence, when the superior oblique muscle is paralyzed, we ought to find a defect at the lower and outer sides of the field of fixation. Such a diminution is to be found (see Fig. 19), but only in very pronounced cases; for the external rectus, by combining its action with that of the inferior rectus, can to a certain degree furnish an equivalent of the abducent action of the superior oblique.

In case of paralysis of the superior oblique muscle, there is a third rotation which the other muscles would not so readily succeed in producing; this is the rotation around the antero-posterior axis, the inclination of the meridians of the eye. It is not shown in the field of fixation, which marks only the excursions of the line of sight, but does not indicate rotations around this line. In order to demonstrate a pathological inclination of the meridians of the eye, it is necessary to have recourse, as Donders has proposed, to accidental images. Thus we know that the superior oblique, while causing the eye to turn downward and outward, exerts upon it a rotation about the antero-posterior axis, which causes the upper end of its vertical meridian to lean towards the nose. The opposite occurs through the influence of the inferior rectus, which inclines that meridian outward. Under normal circumstances these two influences counterbalance, so that when merely looking downward, the eye executes no rotation of this sort, its vertical meridian, for instance, remaining vertical. Withdrawn, however, from the action of the superior oblique and abandoned to that of the inferior rectus, the globe will undergo the rotation which the latter tends to exert upon it; its vertical meridian will lean towards the temple. This inclination will manifest itself directly by the accidental image of a vertical

line. Let the diseased eye be made to fix upon a red ribbon that is stretched vertically across a white background. After a minute, let the eye look downward, then the accidental (green) image of the ribbon will appear on the background no longer vertical, as would be the case if the experiment were made with a normal eye, but inclined towards the temple when there is paralysis of the superior oblique muscle, and towards the nose when it is the inferior rectus that is paralyzed.

It is true that the experiment with the accidental image requires, on the patient's part, intelligence and ability to observe correctly, upon which we cannot always depend in practice. It is, however, in connection with an examination of the field of excursions and some other symptoms with which we shall become acquainted, a most welcome aid when an exact diagnosis without the collaboration of the other eye is to be made.

The diagnosis of paralysis of an ocular muscle is much easier when the fellow-eye is normal. In such a case, when the patient's attention is directed to a distant point, only the better eye will be directed towards that point and kept in that direction by the equilibrium of its muscles. On the contrary, the diseased eye will be abandoned to the traction of the antagonist or antagonists of the paralyzed muscle. They exercise a preponderant action upon the eyeball, and cause its line of sight to deviate in a direction opposite to that in which the affected muscle would have moved it.[1] Such a deviation is called *strabismus,—paralytic strabismus* in this case, in which the condition is due to paralysis of a muscle of the eye.

As an example of this let us take the *paralysis of the external rectus.* Under normal circumstances, this muscle turns the eye towards the temple. Deprived of its action, the eye deviates towards the nose.

Let $O$ (Fig. 2) be a point infinitely distant, so that, in order to fix the eyes upon it, they ought to be directed parallelly forward. In the case of paralysis of the *external rectus* of the left eye, this eye undergoes rotation

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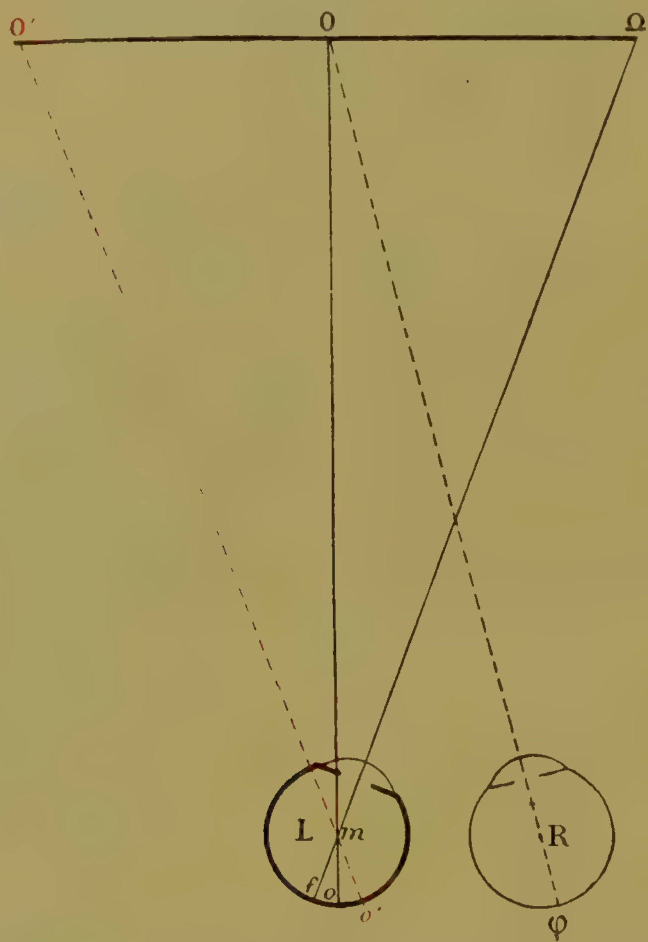
[1] It is important to bear in mind that the antagonist of an ocular muscle is not simply the muscle which causes the eyeball to turn about the same axis, but is the aggregate of the motor forces which act as opponents. Only the external and internal recti can be regarded as pure antagonists, so that, if one of them is paralyzed, the eye's direction and position are the same as if the other had contracted. But even in such a case the other muscles, which act in the same sense, add their influence to that of the antagonist.—The superior and inferior recti are not antagonists any more than are the two obliques, in the sense that, withdrawn from the action of one of them, the eye would behave as if it were abandoned to the other muscle of the same pair. For example, the superior oblique turns the eye downward and outward, and inclines the vertical meridian towards the median plane. Its so-called antagonist, the inferior oblique, turns the eye around the same axis, in the opposite way,—that is to say, upward and outward,—exerting a temporal inclination upon the vertical meridian. The real antagonists of the superior oblique (those to which the eye submits in case it is paralyzed) cause the eye to turn upward and inward, while they incline the vertical meridian towards the temple. This law, which results from simple reflection, is confirmed by observation, as will be seen in another section.

around its centre of motility ($m$) towards the nose; its visual line, instead of being directed towards $O$, will be directed towards $\Omega$.[1] It will form,

FIG. 2.




Convergent strabismus of the *left* eye due to *paralysis of the external rectus* muscle.

with the direction that it ought to have, the angle $Om\Omega$, which is called the *angle of strabismus*.[2]

The visual line of the diseased eye will cut that of the normal eye on the proximate side of the fixation-object. The two visual lines converge, whence the name *convergent strabismus* is given to that form of deviation of the eye.[3]

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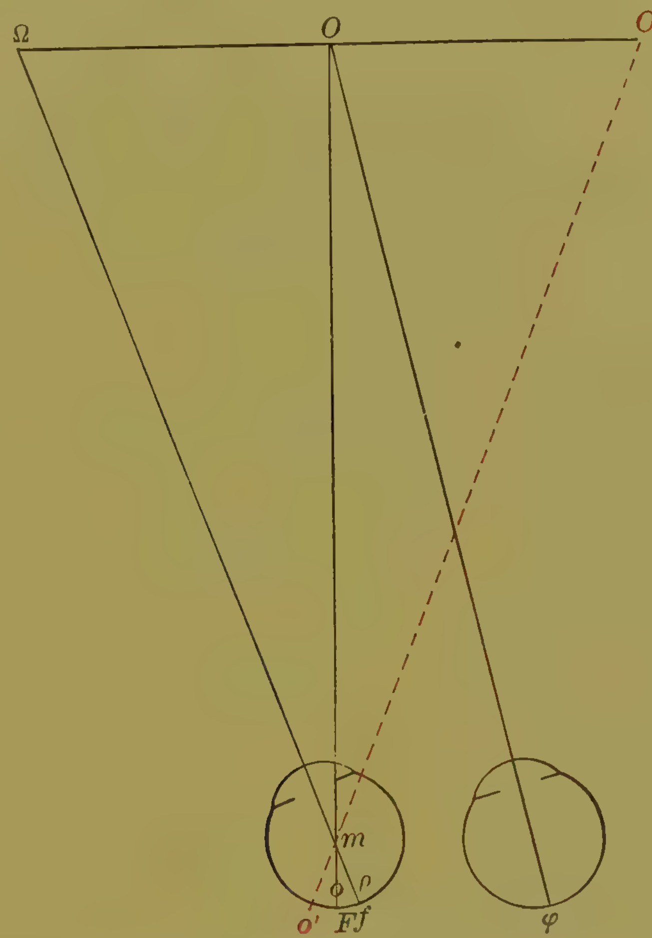
[1] We do not ignore the fact that the optic axes, with reference to which the retinal images are projected, do not cross at the centre of motility, but in the optical centre, or in the nodal points of the eye. (See Landolt, The Refraction and Accommodation of the Eye, pp. 86 and 121.) For the sake of simplicity, we may be permitted to consider these points as fused in one. The resultant error has no effect on the practical questions which are now being treated. For the same reason the visual line and the line of sight may be considered identical, though they are really distinct. (Landolt, loc. cit., p. 117.)

[2] Landolt, Graefe und Saemisch, iii. S. 325, 1874; Landolt, Manual of Examination of the Eyes, p. 50; De Wecker et Landolt, Traité complet, i. p. 909.

[3] It will be noticed that we have supposed the fixation-point $O$ to be at an infinite distance. In this case, the visual line of the right eye is parallel with the direction $oO$, that the left eye ought to have. Again, the distance of the fixation-object being infinite, the few centimetres between the two eyes may be disregarded with reference to it, and we may consider the two eyes as fused into one. In this case, the line $oO$ coincides with the line $\varphi O$, as in Fig. 4, in which we have supposed the two eyes to be superposed in profile.

In the case of paralysis of the *internal rectus muscle*, whose normal function is to direct the eye towards the nose, there will be an opposite deviation,—towards the temple, $Om\Omega$ (Fig. 3),—*divergent strabismus*, relatively to the other eye.

FIG. 3.




Divergent strabismus of the *left* eye due to *paralysis of the internal rectus* muscle.

The action of the *superior oblique* is to turn the eye downward and outward, and to produce, moreover, a rotation around the antero-posterior axis in such a way as to incline the vertical meridian towards the median plane. In paralysis, therefore, of the superior oblique, the eye will be turned upward and inward, and its vertical meridian will lean towards the temple,—*strabismus sursum vergens*, convergent and temporal inclination.

Pathological deviation of the eye produces another of the most characteristic symptoms of muscular paralysis, at least when binocular vision has been present. This is double vision, or *diplopia*.

The explanation of this phenomenon is most simple.[1] Let us again take the example of paralysis of the external rectus of the left eye, and

[1] This explanation of diplopia, with a descriptive figure, we have given in Ann. d'oculistique for 1875, and in our Manual of the Examination of the Eyes, p. 53, 1879. It is also spoken of on p. 822 in vol. iii. of the Traité complet d'Ophthalmologie of De Wecker and Landolt. Nevertheless the authors of manuals continue to copy from each other a totally erroneous figure, which fact serves well to show how greatly this simple phenomenon is still misunderstood.

suppose that the individual's attention is directed upon the point $O$, Fig. 2. The normal eye ($R$) only will be directed towards this point, so that the image of $O$ will be formed upon its fovea centralis, $\varphi$.

In the left eye ($L$), on the contrary, this eye being deviated towards $\Omega$, the image of $O$, instead of being formed on the fovea centralis, $f$, will fall on the point $o$, at the inner, nasal side of the fovea.

Without taking into account the wrong direction of the left eye, the individual projects this image—that is to say, he supposes the object that has caused it to be—at the place where an object ought to be in order that its image, in the normal direction of this eye, will be formed at $o$.

To know where the patient sees the object $O$, we need, therefore, only to cause the left eye to take its normal direction; in other words, to place its fovea centralis opposite the point $O$. The point $f$ will then be where $o$ was before, and $o$ will have passed along to $o'$. Let a straight line be drawn from $o'$ through $m$ to the plane in which is the object $O$ (and in which the patient usually projects the false image), and we shall find at $O'$ the place where the object $O$ appears to be to the deviating eye.

We see that the false image, as we may call it, is at the opposite side from the deviation,—that is to say, at the side towards which the paralyzed muscle would normally direct the eye.

Speaking with relation to the sides of the body, we may say that, in the paralysis of an abductor, the false image appears at the side of the affected eye; at the left, in the example taken, since the external rectus of the left eye turns that eye towards the left.

If the image as seen by the left eye is at the left, and that belonging to the right eye is at the right, the diplopia is called *homonymous*. This characterizes *convergent strabismus*.

The same thing occurs in the case of paralysis of the internal rectus muscle of the left eye. For instance, in Fig. 3, the eye is deflected outward ($fm\Omega$). The image of $O$ is produced at $o$. Restoring the normal direction to the eye, $o$ goes towards $o'$, and $O'$ designates the place at which the paralyzed eye sees the object $O$. Again, it supposes the image to be in the direction opposite to that of the strabismus,—that is to say, in the direction in which the affected muscle would act if not paralyzed.

At the same time, we notice that, in such a case of deviation towards the affected side, the false image ($O'$) is at the side of the sound eye. Since this sound eye sees the object in its real position $O$, the double images are *crossed*, that of the left eye being at the right and that of the right eye being at the left. Thus, crossed diplopia shows divergent strabismus, just as homonymous diplopia expresses convergent strabismus.

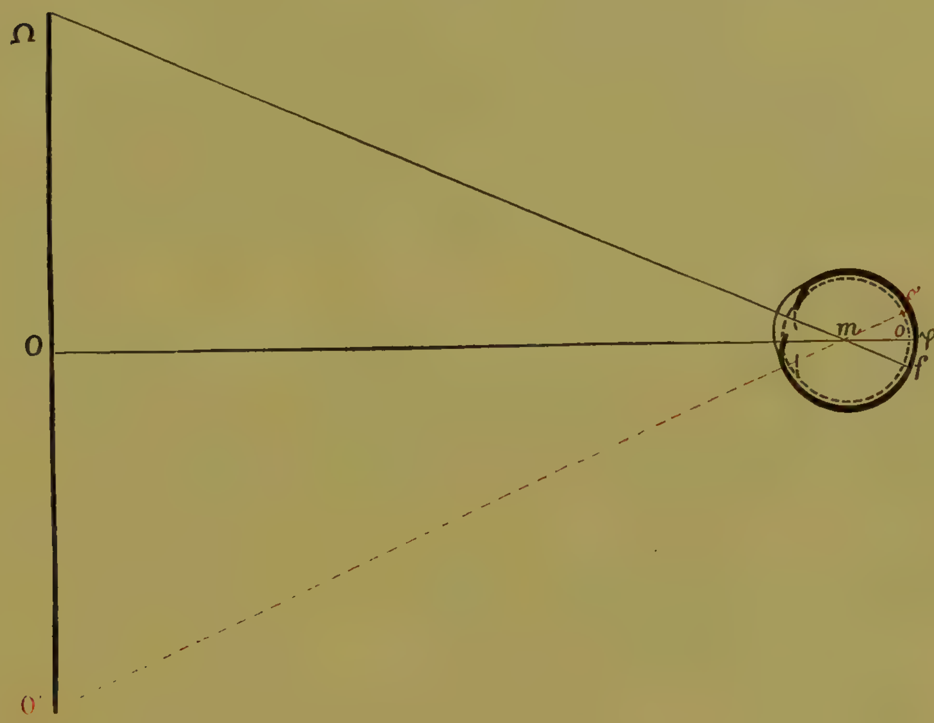
Analogous reasoning gives us the position of the false image in the case of deviation in the vertical direction.

In Fig. 4 the two eyes are supposed to be superimposed in profile. The normal eye (dotted) is directed straight forward towards an object $O$, whose image is formed at the fovea centralis $\varphi$. The diseased eye (plain line)

inclines upward towards $\Omega$ (*strabismus sursum vergens*). The diseased eye receives the image of $O$ at a point $o$ above its macula. Normally, this point would have been struck by rays coming from below the horizon.

Indeed, if we turn this eye back to its normal direction, so that its

FIG. 4.




fovea centralis is opposite $O$, $o$ will take the position of $o'$, and the line $o'mO'$ will give at $O'$ the false image as far below the object $O$ as the visual line of the erring eye was deviated above.

The same figure, inverted, gives the explanation of the diplopia in the case of deviation downward (*strabismus deorsum vergens*) of one eye, in which there is produced, necessarily, the same false projection as in the upward strabismus, only in the inverse sense.

Finally, let us suppose that the eye, instead of having been deviated by rotation around the vertical axis or the transverse axis, has undergone a pathological rotation around the *antero-posterior axis*, so that, for instance, the vertical meridian of the retina is inclined outward, towards the temple.

If this happen to the *left* eye, as shown in Figs. 5*a* and 5*b*, which illustrate the condition of the two eyes as recognized from behind, the visual disturbance which results from it can be easily determined. The image $yx$ of a vertical line will coincide in the normal, the right eye ($R$) with the vertical meridian ($VV$) of the retina. In the left eye, whose meridian $VV$, which ought to be vertical, leans outward, the image $yx$ will make an angle with that meridian. Hence the object will seem to be inclined, in its turn.

In order to determine the direction of the inclination, we suppose the eye to have resumed its normal position. (Fig. 5c.) Then the meridian *VV* of its retina, which is normally vertical, becomes vertical, and the

FIG. 5a.

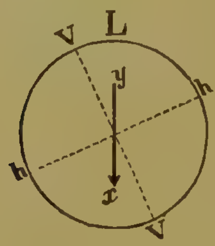



FIG. 5b.

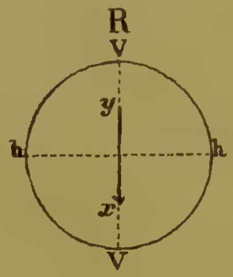



FIG. 5c.

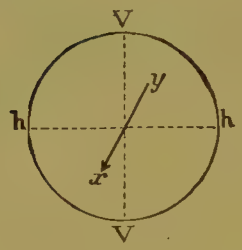



FIG. 5d.


upper extremity of the image ($y$), which corresponds to the lower extremity of the object, is situated to the inner (nasal) side. Hence the image of this point will be seen to the outer side, towards the temple. ($Y$, Fig. 5d.) The lower end ($X$) of the image, which falls on the temporal part of the retina, will be projected inward—that is to say, towards the nose ($X$, Fig. 5d) —and the object will seem to be inclined, as is shown by the line $XY$.[1]

[1] This false image is not to be confused with the projection of the accidental image. The accidental image is only the vertical meridian of the eye marked by a line whilst the meridian was in its correct position (only the affected eye fixing). When the eye turns and

It will, without explanation, be seen that here, again, the degree of apparent inclination of the image is equal to that of the pathological rotation of the eye.

Hence it must be understood that the false image is always the inverse of the direction and the position of the paralytic eye. With a nasal (internal) deviation, the false image is temporal (external); or again: convergent strabismus is accompanied by homonymous diplopia.—With a temporal (external) deviation, the false image is nasal (internal); therefore divergent strabismus is characterized by crossed diplopia.—In upward deviation (strabismus sursum vergens), the false image is inferior.—In downward deviation (strabismus deorsum vergens), the false image is superior. —Pathological temporal inclination of the eye produces nasal obliquity of the image, and a median (nasal) inclination of the eye temporal obliquity of the image.

Since the false image is always situated in a position and direction contrary to those of the diseased eye, and the deviation is the contrary to the direction which the eye takes when it is normally acted upon by the muscle, the false image must correspond, in all respects, with the direction which the muscle normally gives to the visual line as well as to the meridians of the eye.

The red line which on the following pages stands for the false image corresponds, therefore, at the same time, to the direction and the inclination that the muscle in question imparts normally to the eye. It is only necessary to substitute the vertical meridian of the eye for the false image, and the pathological scheme will have been transformed into a physiological one, exhibiting the normal action of the ocular muscles.

The diplopia which results from the paralysis of an ocular muscle necessarily gives great annoyance to the patient. He is troubled in making his way about. If he sees two objects instead of one, he is not certain which is the true one. If the double images are partly superimposed, whether because the paralysis is not complete or because the object is too large or too near, the latter appears deformed. To obviate these and other inconveniences with which we shall later become acquainted, the patient closes one eye, thereby sacrificing binocular vision. Sometimes he has recourse to another plan: he substitutes a rotation of the head for the defective rotation of the eye.

If, because of paralysis of the left external rectus muscle (Fig. 6*a*), the patient cannot turn the corresponding eye towards the temple, he will turn his head in the same situation, until the visual line of his left eye (*LX*)

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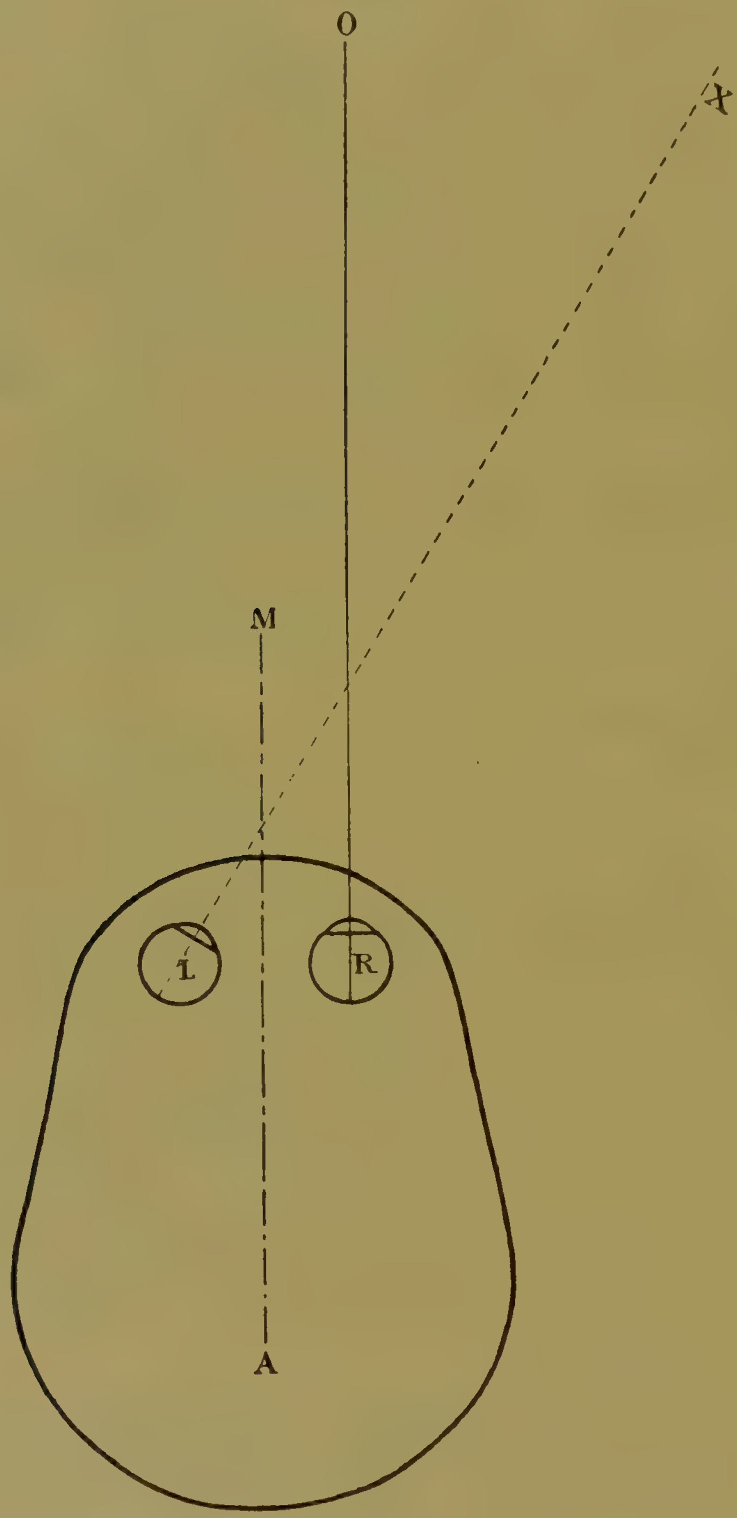
the meridian inclines, the image turns with the eye and is projected with the same lateral inclination that the meridian has assumed.

The false image corresponds to the projection of an image which the eye receives while it is in an incorrect position. Hence it is projected in the opposite direction from the inclination of the meridian of the eye (Fig. 5*d*),—that is, in the inverse direction of the pathological rotation of the eye.

is directed towards the object ($O$) that is fixed by the right eye. (Fig. 6*b*.) He thus sees the object binocularly, and the diplopia will disappear. In

FIG. 6*a*.




other words, instead of turning the eye in the head, he causes it to turn with the head.

If one of the patient's eyes be directed upward, in consequence of a paresis of a depressor muscle, he will turn his head downward. It is the head that will make the movement which the eye is incapable of executing.

If, on the contrary, there is a deficient amount of motility upward (*strabismus deorsum vergens*), he will direct his head upward.

If his eye has undergone a pathological rotation around its antero-

Fig. 6*b*.

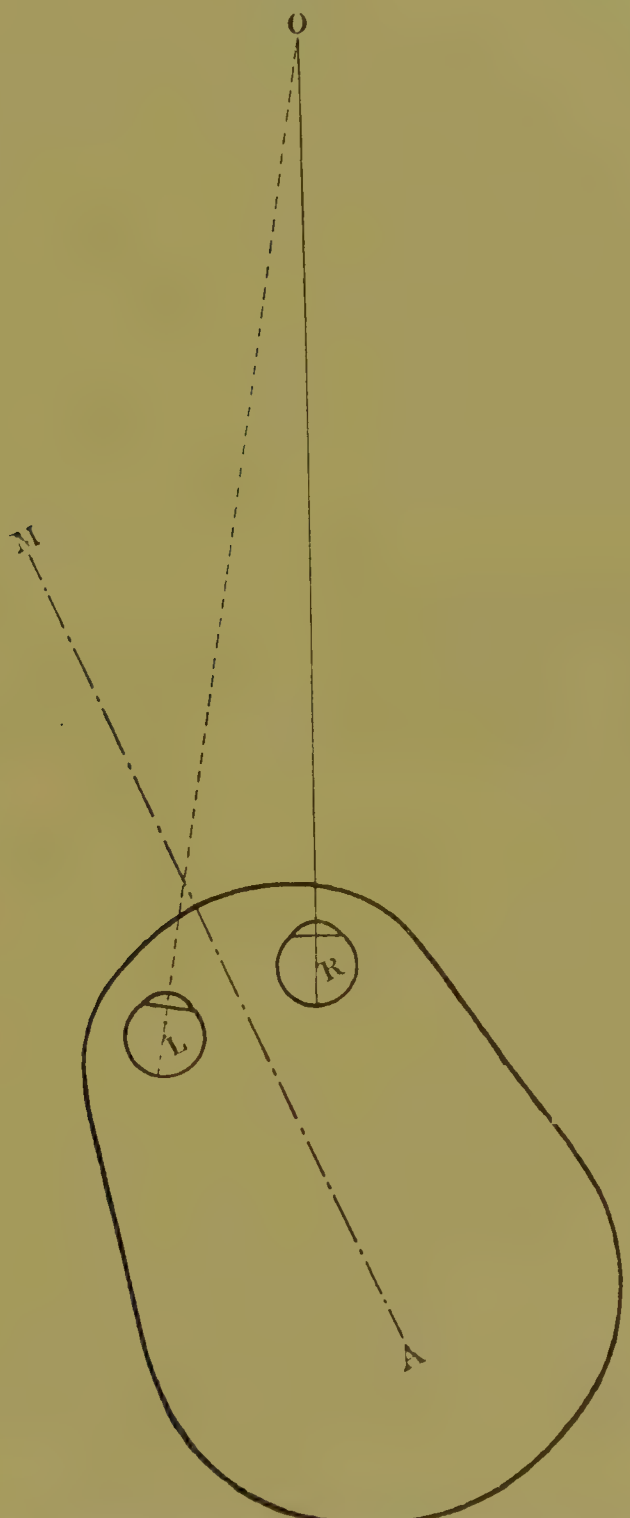



Diagram illustrating the movement of the head to obtain single vision with the two eyes in paralysis of the left external rectus muscle.

posterior axis in such a way that its vertical meridian is inclined towards the left (Fig. 5*a*), the patient will lean the head in the inverse direction until the meridian of his eye, which ought to be vertical, really becomes so anew. (Fig. 5*c*.)

Finally, if one of the patient's eyes has undergone a pathological rotation at once upward, inward, and around the visual line to the left, he will direct his head downward and outward, and will incline it towards the right side.

Hence it will be seen that the corrective direction of the head is opposite to the direction and position of the paralyzed eye, and is identical with the projection of the false image. The patient turns his face towards the false image and inclines his head as the latter is inclined. Indeed, the false image corresponds to the direction and to the inclination which the paralyzed muscle, when normal, exerts upon the eye; and it is to make good the lost action of the muscle that the patient turns his head.

The colored lines on the following pages may claim, therefore, a third meaning. They not only show the direction and position assumed by the eye under the influence of the normal contraction of the muscle, as well as those of the false image in the case of paralysis of the muscle, but they indicate also the direction and corresponding corrective position of the head.

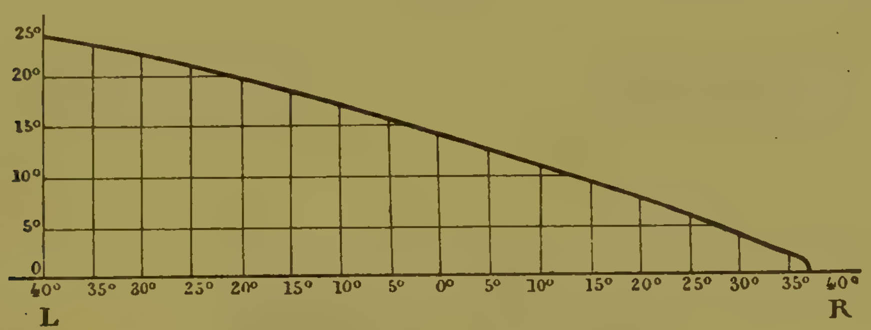
It has been seen that the patient can put an end to the diplopia, bring about the fusion of the double images, and direct both his eyes simultaneously upon the fixation-object, by turning his head in the direction opposite to his strabismus. He ought to be able to secure the same result by keeping his head still and displacing the object in the direction in which his eye is deviated. Let us consider Fig. 6*b*. It is necessary only to turn the diagram till the median line *MA* is directed forward. Then the object *O* will be at the right, and will be as readily fixed by the normal right eye as by the left eye, whose external rectus is paralyzed. This is what really happens. The diplopia diminishes in proportion as the fixation-object moves in the direction of the deviation of the eye. On the contrary, the distance between the two images increases, when the object moves towards the side of the paralyzed muscle. The two images separate more and more from each other; the false image seems to move from the object in the direction of the paralyzed muscle. In the first case, the object enters more and more into the domain of the muscles that are acting normally and are capable of associating their action with that of the muscles of the sound eye. On the contrary, in the second case, the object enters into the sphere of action of the paralyzed muscle, whose defect makes itself felt increasingly in proportion as the object is carried farther in that direction.

Hence the angle of strabismus—that is to say, the separation of the visual lines of the two eyes—has nothing absolute about it; it may become *nil* in the direction of the deviation, while it increases in the direction of the paralyzed muscle.

In order to avoid confusion, the term *angle of strabismus*, properly so called, is used to designate the angle that is formed by the lines of sight when the healthy eye is directed straight forward towards an infinitely distant object.

The domain within which the lines of sight of the two eyes may meet in the same point of fixation—in other words, the *domain of the fusion of the two images*—is more limited in proportion as the paralysis is more nearly complete. This domain of binocular single vision is separated from the domain of the double vision by a fairly distinct line of demarcation for the paralysis of different ocular muscles.

FIG. 7.

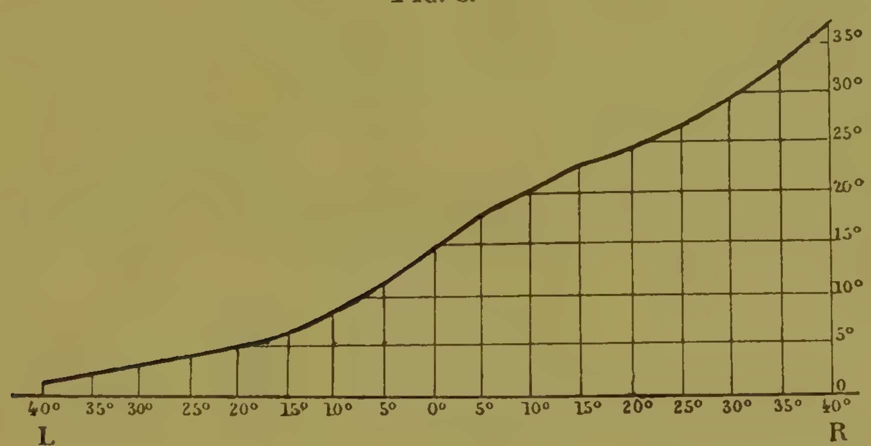



The curve in Fig. 7 represents the diplopia of a patient afflicted with paresis of the *left external* rectus, having produced a convergent strabismus of 14 degrees.

The numerals along the horizontal line indicate, in degrees, the excursions of the eyes from the zero-point, which corresponds to the primary position,—that is to say, looking straight ahead. The numerals on the vertical line indicate the degrees of the strabismus and of the diplopia.

It will be seen that, when the patient directs his eyes towards the right, at an angle of 37°, the diplopia disappears in consequence of the fusion of the double images. On the contrary, when he looks towards the left, the diplopia increases. It amounts to 14° when he looks straight ahead, and to 25° when he looks forty degrees in the direction of the paretic muscle.

FIG. 8.




The curve in Fig. 8 represents the diplopia of a patient having paresis of the *right external* rectus. In this case, the diplopia extends over the entire field of excursion. At an angle of 40° to the left, the two images are still separated by two degrees; then that of the right eye disappears, because the nose hides the object from that eye. At the same angle to the right, the diplopia amounts to 35°, and it is then the image belonging to the left eye that disappears behind the nose. In the primary position, the diplopia, and, consequently, the strabismus, amount to 15°.

The domain of fusion, the *field of binocular fixation* of the paralytic eye, is, moreover, in no wise equal to the common part of the superimposed fields of fixation of the two eyes. It is much more restricted than is the latter.

Thus, Fig. 9*a* represents the horizontal extent of the excursions of the *left* eye of one of my patients having *paresis of the external rectus*, with a

convergent strabismus of 8°. This field of fixation is limited on the outer side, where it reaches only 22°, while its nasal limit shows no alteration, measuring as much as 48°.

The excursions of the *right* eye are normal. They go to 48° on both sides. (Fig. 9*b*.)

In Fig. 9c the two fields are superimposed. It is evident that the zone which is common to them is quite extensive. It goes from 22° at the left

Fig. 9*a*.

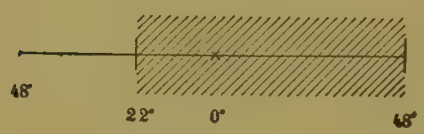



Fig. 9*b*.

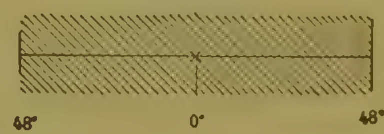



Fig. 9*c*.


to 48° on the right. But examination of the binocular vision demonstrates that the fusion of the two images commences only at an angle of 33° to the sound side. Hence the field of binocular fixation in the horizontal meridian has an extent of scarcely 15°, instead of the 70° which are common to the excursions of the two eyes in this meridian. In other words, if the two eyes command this space with the excursions of their lines of sight, they do not command it *simultaneously*.

Still, this fact has nothing surprising about it. If, in a case of paresis, the sick eye can still move to a certain degree in the opposite direction from its deviation, this movement requires of the enfeebled muscle, necessarily, a much greater effort than if it were normal, and more than it does of the associated muscles of the other, the healthy eye. Hence we need not be astonished if the two eyes succeed only with difficulty in associating their movements, even in the space which each, by itself, commands.

The abnormal effort which the enfeebled muscle is obliged to make when it tries to move the eye, gives rise to still other very important symptoms.

Let us resume consideration of Fig. 2, which represents a case of paralysis of the external rectus of the left eye, and suppose that the other eye is excluded from vision, being blind or covered by a diaphragm. The patient wishes to fix the object *O*,—that is to say, to give his eye, *L*, such a direction that the image of *O* may be formed on its fovea centralis, *f*. If the paralysis of the abducens be complete, there is nothing left for the patient to do but to turn his head towards the left, through an angle equal to the deviation of the eye, or to carry the object to the right, until it is on the visual line *f Ω*.

But suppose there is only *paresis*, and that the external rectus has re-

tained enough contractility to direct the eye towards the object. This rotation, executed by a paretic muscle, necessitates a stronger nervous impulse in proportion as the muscle is weaker.

Now, it is by the aid of the muscular sense that we make our way about, and particularly by the aid of the *sense of the ocular muscles.*

The patient affected with *paresis of the left external rectus* will, then, suppose the object fixed to be so much the more to the *left* side, as he has brought more energy into play in order to reach it with the visual line.

If, guided only by the paretic left eye, he hastens towards an open door, he runs the risk of a collision with the left side of the door-frame. Hence the very characteristic *gait* of such a patient: instead of going straight towards the point of destination, he at first goes too much to the left, and it is only later, on perceiving his error, that he rectifies his course, often betaking himself suddenly to the opposite side, where the object actually is. For the same reason, he pours water to the left side of the glass; instead of dipping his pen in the inkstand, he puts it to the left side of the stand, etc.

This FALSE PROJECTION, as it is called, necessarily takes place always in the direction of the normal action of the paretic muscle, exactly like the projection of the false retinal image which gives rise to diplopia.

However, the two phenomena must not be confounded with each other. They are not at all identical. *Diplopia* is produced even when the eyes are at rest in their position of equilibrium, and results, as we have explained, from the comparison of the place in the retina where the image is formed in the healthy eye, with the one where it is produced in the deviated eye.

*False muscular projection* does not come into play until the moment when an effort is demanded of the paretic muscle. If, instead of directing the deviated eye towards the fixation-object, the patient displaces the latter or turns his head so that its image is received on the fovea centralis without any effort of the affected muscle, he will not be deceived as to the position of the object. Thus, the false projection *diminishes* in the direction of the *deviation,* while it *increases* in the direction of the *paretic muscle.*

Hence this pathological phenomenon to which the paralysis gives rise follows also from the physiological action of the muscle. To a person one of whose abductors is paralyzed, the ambient world will seem displaced towards the affected side.—If it be the internal rectus that is paretic, the false projection will be towards the healthy side.—In the same way, the hand will seek an object below its real position, and at the temporal side of where it really is, when the patient fixes only with an eye affected with paresis of the superior oblique.

The false projection becomes especially manifest, when one asks the patient to tap rapidly with his finger an object located on the side of the paralyzed muscle.

In order that this experiment may succeed, it is indispensable to hide from the patient the hand with which he is to touch the fixation-object. Otherwise, the image of the hand being formed on the same retina with that of the object, he will rectify the false muscular projection by the correct retinal projection. The simplest means of succeeding in this, is to place a large piece of cardboard between the patient's neck and the wall on which the fixation-point has been marked. The patient is then to direct his index finger, under the cardboard, towards the place where he supposes the point, fixed by his paretic eye, to be.

Donders advises that the patient's eye be closed while he points out the location of the fixed object. It is easy to convince one's self that, under these circumstances, a healthy person will always point correctly, while the ill one will point wrong and too far in the direction of the paralyzed muscle.

Let us see, now, what the *sound* eye is doing while the ill one is fixing. This is easy; for, in order to exclude the sound eye, we need not cover it entirely, provided we hide the fixation-object from it. We thus learn that *the healthy eye is deviated in the direction in which the paretic muscle turns the affected eye.*

For instance, in a case of paresis of the *external rectus* of the *left eye*, the right eye squints towards the *left* when the left eye fixes alone. If the *left* (fixing) eye be the victim of *paresis* of the *superior rectus*, the right eye is directed *upward* and to the *right*,—that is to say, towards the side to which the paretic muscle would have directed the left eye.

Finally, if the paretic muscle turns the eye around its antero-posterior axis, *inclining the vertical meridian*, for instance, towards the *nose*, as does the superior rectus, the sound eye will execute an analogous rotation (towards the *temple* in this example) when the affected eye alone is fixing.

The deviation which the healthy eye undergoes, while the paretic eye is fixing, is called SECONDARY DEVIATION. It may be considered as the effect of the paretic muscle acting upon the healthy eye.

In order to represent the direction and inclination assumed by the healthy eye while the ill eye is fixing, one need only consider the red lines of our special part (pages 22–28), but interchanging the letters. For instance, in case of paresis of the *left* eye, the letter *R* will be substituted for *L*, and the red line will represent the secondary deviation of the right eye while the affected left eye fixes. This is, therefore, a *fourth* signification which the accompanying diagrams may claim.[1]

The secondary deviation is especially striking when, the fixation-object and the patient's head being immobile, each eye is successively covered and uncovered. The deviation then manifests itself not only directly to the

[1] The diplopia resulting from the secondary deviation is directly deducible from the position of the sound eye. The image corresponding to it is, in all points, the inverse of this position.

view, but also by the *correcting movement* that the eye is forced to make in order to direct itself towards the fixation-object.

On making this experiment carefully, one sees a phenomenon which is one of the most important for the diagnosis of paralysis of the ocular muscles. It is that the *secondary deviation* (of the healthy eye) is always *more extensive than the primary deviation* (of the paretic eye).

An intelligent patient readily perceives that, when he fixes with the affected eye, the diplopia and apparent inclination are considerably stronger than when he fixes with the healthy eye.

This phenomenon is explicable as follows. The movements of the eyes are associated; one of them cannot be moved unless the other is. If one of them goes from right to left, for instance, the other follows it, so that the internal rectus muscle of the right eye may be considered the associate of the external rectus of the left eye. Under normal conditions, therefore, the two always receive the same nervous impulse.

Now, if, in consequence of paresis, the left external rectus claims a stronger nervous impulse than usual, in order to direct itself towards the object fixed, the intact internal rectus of the other eye will respond to this surplus of stimulation by a surplus of rotation, which it will communicate to the eye to which it is attached. The right eye will be turned inward more than the left one is, when the right eye fixes. Indeed, the left eye squints only because it abandons itself to the preponderance which the internal rectus assumes over the enfeebled externus, but not in consequence of an active contraction of the former.

The same is true in the case of the superior rectus. If that of the left eye be paralyzed, and we oblige the patient to use that eye alone for vision, he will bring into play more than normal innervation, in order to direct the eye in the sense of the paretic muscle—that is to say, upward and towards the nose—and to counterbalance the effect of its antagonists. The same effort will turn the healthy eye upward and slightly outward, more than it does the affected eye: so that the difference in height as well as the divergence will be much more marked during the fixation of the affected eye than during that of the healthy eye. The same thing occurs in the case of rotation around the antero-posterior axis. The effort which the affected eye makes to restore its vertical meridian leaning towards the temple, will impose upon the sound eye a much more marked inclination towards the temple on its side.

The case is not rare wherein one is uncertain not only as to which of the eyes is the affected one, where there is paresis of an abductor or an adductor, but also as to which of the levators or depressors is paretic, and to which eye it belongs. It even happens that, the visual value of the two eyes being very different, it is the better of the two which is the victim of muscular paresis. In such a case, the patient continues to use that eye for fixation, and the other deviates. One is then the more tempted to consider the latter as the paralytic eye as its deviation is more marked. More than

once patients have come who have, for months, had electricity applied to the healthy eye instead of to the affected one.

The examination of the field of fixation and the study of diplopia, but especially that of the secondary deviation, will guard us against such an error.

Moreover, we shall become acquainted with a form of strabismus wherein the deviation is sensibly the same for both eyes. The secondary deviation will help us to distinguish the two kinds of strabismus one from the other. Hence it constitutes, from more than one point of view, a most precious means of diagnosis.

We have demonstrated[1] that *the difference between the secondary and primary deviations is equal to the angular value of the false projection.*

If, for instance, the patient has convergent strabismus amounting to 8°, in consequence of paresis of the left external rectus, and if, when fixing with the affected eye, there is present a secondary deviation of 12°, he will suppose the fixation-object to be $12° - 8° = 4°$ to the left of its real location.

There is nothing surprising about this fact: both phenomena are due to the same cause. Each is the expression of the surplus of innervation required by the paretic muscle. This excess of nervous impulse provokes an excess of deviation of the *sound* eye, whilst it manifests itself, on the affected eye, as an error of orientation, as false projection.

## RÉSUMÉ OF THE GENERAL SYMPTOMATOLOGY OF PARALYSIS OF THE OCULAR MUSCLES.

We have become acquainted with a series of phenomena to which paresis of an ocular muscle gives rise, and we have seen that they may all be reduced to the action of that muscle upon the eyeball in normal circumstances. These phenomena are produced either in the same direction in which the muscle turns the eye, or in the opposite direction.

Let us arrange these phenomena in two columns, and we shall have our symptomatology reduced to its simplest expression.

There are produced—

| In the sense of the *physiological action* of the muscle: | In the *opposite* sense from the physiological action of the muscle: |
|---|---|
| (Red lines of special part ) | |
| 1. The *defect of the excursion*,—limitation of the field of fixation. | 2. The *deviation of the eye*,—the strabismus. |
| 3. The *false image*,—increase of diplopia. | 4. The *diminution of the diplopia*,—the fusion. |
| 5. The *direction of the face.* | |
| 6. The *false projection.* | |
| 7. The *secondary deviation* of the sound eye. | |

[1] Landolt, Archives d'ophthalmologie, Mai, 1893.

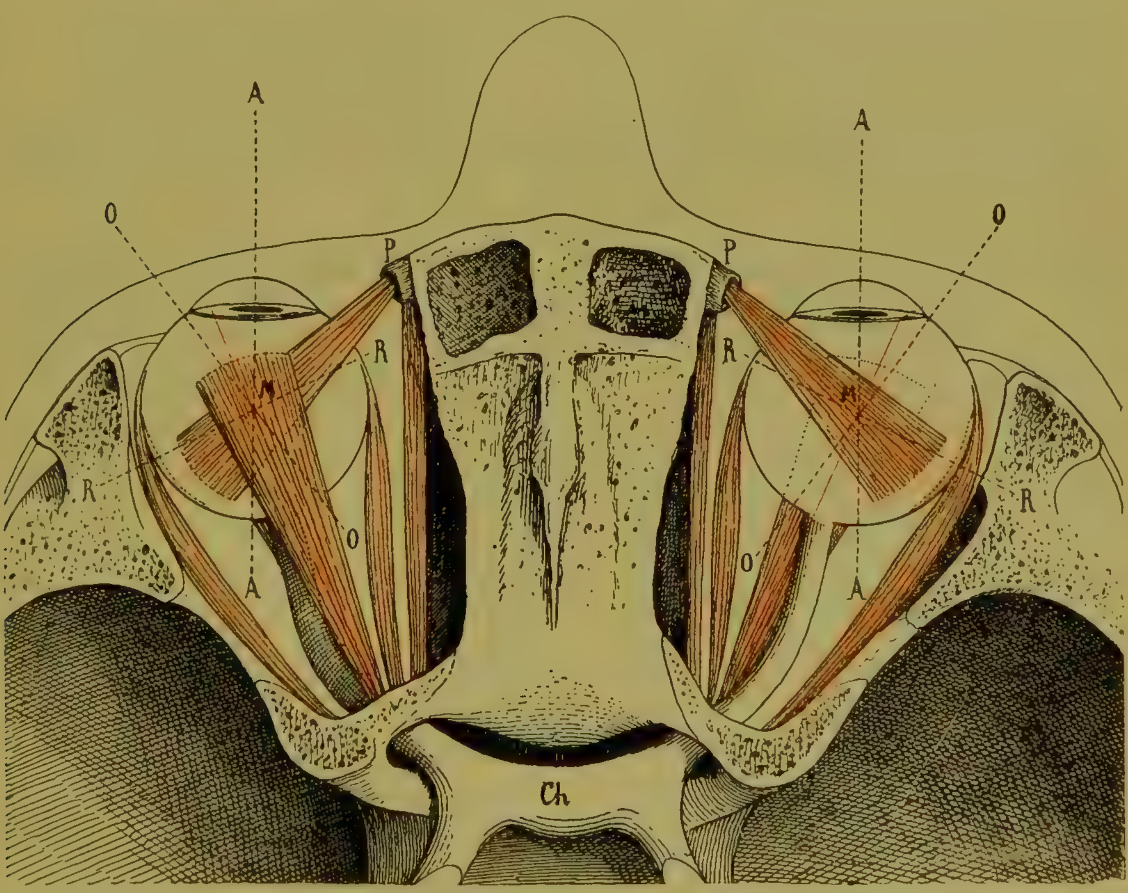
## SPECIAL PART: SYMPTOMS OF PARALYSIS OF THE MOTOR MUSCLES OF THE EYE.

When we bear in mind the anatomy and physiology of an ocular muscle, nothing is easier than to find, by the help of the rules that we have just laid down, the phenomena to which its paresis must give rise. We shall, therefore, for each of the muscles preface the symptomatology by a brief anatomical[1] and physiological *résumé.*

### EXTERNAL RECTUS.

The *external rectus* takes its origin at the optic foramen, is directed forward and outward, and is inserted 7 millimetres from the temporal margin of the cornea. (Fig. 10 and Fig. 17, *EE.*) The muscular plane is

FIG. 10.




*M*, centre of motion; *AA*, antero-posterior axis; *RR*, axis of rotation of superior and inferior *recti*, forming an angle of 63° with *AA*; *OO*, axis of rotation of superior and inferior *oblique* muscles, forming an angle of 39° with *AA* (the red lines, perpendicular to the axis of rotation, indicate the corresponding *muscular planes*); *P*, pulley (trochlea) of superior oblique muscle. The rectus superior of the right eye has been removed. The insertion of the inferior oblique is rarely visible from above; it can be seen better from the temporal side (Fig. 17). *Ch*, chiasma.—*E. Landolt del.*

horizontal, the axis of rotation vertical; hence the external rectus turns the cornea strictly towards the temple, without producing any inclination of the vertical meridian.

**Paresis of the External Rectus.**—The excursion of the eye is limited towards the temple,—that is to say, towards the side of the affected eye.

[1] Fuchs, Archiv für Ophthalmologie, xxx., 4, S. 1 *et seq.*

The eye is deviated towards the healthy side,—*convergent strabismus.*

The false image is on the same side with the affected eye (*homonymous diplopia*), is parallel to that of the other eye, and on the same level with it.

The diplopia increases towards the side of the affected eye, and diminishes at the side of the healthy eye.

The false projection takes place towards the affected side.

The face is directed towards the side of the affected eye.

The secondary deviation of the healthy eye is towards the affected side

FIG. 11*a*.

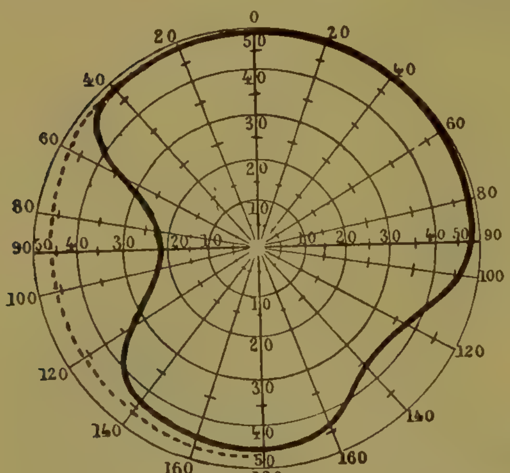

Relative position of the double images in *paralysis* of the *external rectus* of the *left* eye.

FIG. 11*b*.


Relative position of the double images in *paralysis* of the *external rectus* of the *right* eye.

(convergent strabismus), and the diplopia is still homonymous, only more pronounced than when the healthy eye fixes.

FIG. 12.




Field of fixation of a *left* eye affected with *paresis* of the *external rectus.* (The dotted line corresponds to the normal field of fixation.)

We give in Fig. 12 the field of fixation of a case wherein paresis of the external rectus has brought about convergent strabismus of 8°.[1]

When an eye affected by paresis of its external rectus reaches the temporal limit of its field of excursion, one often sees it oscillate upward and downward by virtue of the contraction of the obliqui which are trying to supply the insufficient abducent action of the paralyzed muscle.

INTERNAL RECTUS.

The *internal rectus* originates at the optic foramen, and is directed forward and towards the median plane, to be inserted 5.5 millimetres from the inner corneal margin. (Fig. 10.) The muscular plane is horizontal, and its axis of rotation vertical. Hence the internal rectus turns the cornea directly towards the nose without inclination of the vertical meridian. The external and internal recti are antagonists.

**Paresis of the Internal Rectus.**—The excursion of the affected eye is limited on the side of the healthy eye.

The eye is deviated towards the temple,—*divergent strabismus.*

[1] Compare also an analogous fixation-field, Landolt, Archives d'Ophthalmologie, 1880, i. p. 605.

The false image appears on the side of the healthy eye; it is parallel with that of the other eye and on the same level,—simple *crossed diplopia.* The diplopia increases towards the side of the sound eye, and diminishes on the side of the affected eye.

The face is directed to the side of the sound eye.[1]

The false projection is towards the healthy side.

The secondary deviation is towards the healthy side (divergent strabis-

Fig. 13*a*.


Relative position of the double images in *paralysis* of the *internal rectus* of the *left* eye.

Fig. 13*b*.

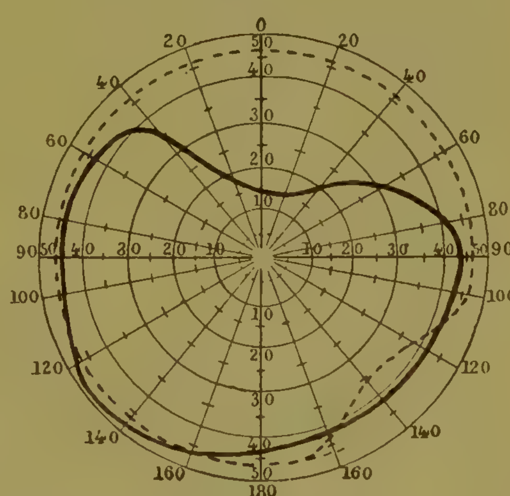

Relative position of the double images in *paralysis* of the *internal rectus* of the *right* eye.

mus); hence the diplopia is still crossed, only more pronounced when the sound eye fixes.

SUPERIOR RECTUS.

The *superior rectus* originates at the optic foramen. (Fig. 10.) It is directed forward, upward, and slightly outward, to be inserted 7.7 millimetres from the upper corneal margin. —The muscular plane is vertical, but forms an angle of 27°, opening outward, with the median plane. (*AA*, Fig. 10.) Its axis of rotation (*RR*, Fig. 10) is sensibly in the horizontal plane, and forms with the antero-posterior axis an angle of 63°, its median extremity directed forward.—The superior rectus directs the eye upward and inward, and inclines the vertical meridian towards the nose.

Fig. 14.




Fixation-field of a *left* eye affected with *paresis* of the *rectus superior.*

**Paresis of the Superior Rectus.** —Excursion defect upward and towards the healthy side. (Fig. 14.)

Deviation of the eye downward and towards the affected side. *Strabismus deorsum vergens*, and slightly *divergent.*

[1] It is to be noticed that a patient whose face is turned towards the left may be affected with paralysis of either the left external or the right internal rectus. Indeed, these two muscles turn the eyes to the left. Hence their paralysis necessitates the same compensatory rotation of the head. But the differential diagnosis in such a case presents no difficulty, since paralysis of the externus produces convergent and that of the internus divergent strabismus.

The vertical meridian is inclined towards the temple.

*Vertical and Slightly Crossed Diplopia.*—The *image* of the *diseased* eye is *higher* than that belonging to its fellow-eye, and slightly *inclined towards* the *nose.* (Figs. 15*a* and 15*b*.)

Fig. 15*a*.

Relative position of the double images in *paralysis* of the *rectus superior* of the *left* eye.

Fig. 15*b*.

Relative position of the double images in *paralysis* of the *rectus superior* of the *right* eye.

The *difference in height between the two images increases* when the patient looks *upward,* and also when he looks *towards the diseased side.* In this case, the line of sight takes the same direction as the rectus superior muscle (or, what amounts to the same thing, is directed perpendicularly to the axis of rotation of this muscle). The latter then becomes a pure levator, and the defect in elevation of the eye, resulting from its paralysis, makes itself so much the more felt.

The inclination of the vertical meridian and that of the false image diminish at the same time.

The inverse takes place when the patient looks towards the healthy side. In this case, the line of sight of the diseased eye approaches the axis of rotation of the muscle, and the latter, instead of raising the cornea, rather turns it around the line of sight. Hence, when the muscle is paralyzed, the action of its antagonists makes itself felt not so much in depressing the eye as by a pathological rotation around the visual line.

Consequently the *difference in height* of the two images *diminishes* when the patient looks towards the side of the *healthy eye,* while the *inclination* of the false image *increases* at the same time.

The *vertical diplopia diminishes* still more when the patient looks *downward.*

The *face* is directed *upward,* and there is a tendency to *incline the head* towards the *shoulder of the healthy side,* in order to obviate the inclination of the false image.

The *false projection* is *upward* and *towards the healthy side.*

The *secondary deviation* of the healthy eye is *upward* and *towards* the

healthy side, with *temporal inclination* of the vertical meridian. Hence there is, in this case, a difference in the height of the two images; that belonging to the healthy eye being the lower, on the opposite side (crossed diplopia), and inclined towards the side of the diseased eye.

### INFERIOR RECTUS.

The *inferior rectus* takes its origin at the optic foramen. (Fig. 10.) It is directed forward, downward, and slightly outward, to be inserted 6.5 millimetres from the border of the cornea, obliquely, its nasal extremity being more advanced than its temporal. The muscular plane, as well as the axis of rotation (*RR*, Fig. 10), is sensibly the same as for the superior rectus.

The inferior rectus turns the cornea *downward* and *inward*, and inclines the vertical meridian *towards the temple.*

**Paresis of the Inferior Rectus.**—Defective motility downward and towards the healthy side.

Eye deviated upward and towards the diseased side. *Strabismus sursum vergens*, and slightly *divergent.* The vertical meridian is inclined towards the *nose.*

The *false image* is the *lower*, is on the side of the *sound eye* (*vertical and slightly crossed diplopia*), and its upper end tends *towards the temple.* (Figs. 16*a* and 16*b*.)

FIG. 16*a*.


Relative position of the double images in *paralysis* of the *inferior rectus* of the *left* eye.

FIG. 16*b*.


Relative position of the double images in *paralysis* of the *inferior rectus* of the *right* eye.

The difference in height between the two images *increases* when the patient looks *downward*, especially when the affected eye is directed *towards* the *diseased* side, because then the inferior rectus becomes a pure depressor and loses its action as a rotator around the line of sight. This is why the inclination of the meridian and, consequently, that of the false image decrease in this case.

On the contrary, the *inclination of the image increases* and the *vertical diplopia diminishes* when the eye is carried *towards the healthy side*, because,

on directing itself perpendicularly to the muscle implicated, the eye undergoes only rotation around its visual line, which then coincides with the axis of rotation of the muscle.

The *vertical diplopia diminishes* when the patient looks *upward* and *towards the healthy* side.—The *inclination of the image diminishes* when the patient looks *towards the affected side.*

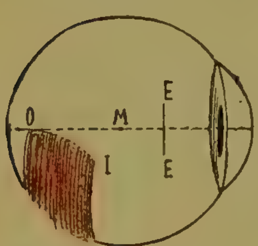
The *face* is directed *downward* and *towards the healthy side*, and slightly *inclined* over the shoulder of the *diseased side.*

*Secondary deviation* of the sound eye : downward and to the healthy side; its vertical meridian leans towards the nose: hence the image which corresponds, under such conditions, to the healthy eye is the higher, on the opposite side (crossed diplopia), and is inclined towards the temple.

## INFERIOR OBLIQUE.

The *inferior oblique* takes its origin from the anterior and inner part of the floor of the orbit, at the margin of the naso-lacrymal canal, and is directed backward and outward, around the eyeball, to be inserted on its posterior, external portion, just under the horizontal meridian. We have indicated this insertion, in Fig. 10, by a little *red* mark; in reality it is not quite visible from above. It presents itself wholly on the temporal side. (Fig. 17.)

Fig. 17.




Right eye seen from temporal side.—M, centre of motion; EE, insertion of rectus externus; OI, insertion of obliquus inferior.

The muscular plane is vertical and forms an angle of about 51° with the median plane. The axis of rotation (*OO*, Fig. 10) is sensibly in the horizontal plane, forming with the antero-posterior axis an angle of 39°, opening outward.

The inferior oblique directs the cornea upward and outward and inclines the vertical meridian outward.

**Paresis of the Inferior Oblique.**—The excursions of the eye are limited upward and to the affected side.

The eye is deflected *downward* and *towards the healthy side. Strabismus deorsum vergens,* and slightly *convergent.*

The *vertical meridian* is inclined towards the *nose.*

The *false image* is the *higher*, and at the side of the *diseased* eye. *Vertical and slightly homonymous diplopia.* The upper end of the *image* leans *towards the temple.* (Fig. 18.)

The *difference in height increases* when the patient looks *upward*, and also when he looks *towards the healthy side*, because, in this case, the line of sight approaches the muscular plane and tends to place itself perpendicularly to its axis of rotation. The inferior oblique thus becomes a pure levator, and the defect in the elevation of the eye, as well as the difference in height between the double images resulting from its paralysis, becomes most marked.

On the contrary, the *inclination of the meridian* and, consequently, that

of the false image are *more accentuated* when the gaze is *towards the ill side;* for, if the line of sight coincides with the axis of rotation of the oblique muscle, the latter turns the eye only around this line of sight, instead of elevating or lowering it.

FIG. 18*a*.

Position of the double images in *paralysis* of the *inferior oblique* of the *left* eye.

FIG. 18*b*.

Position of the double images in *paralysis* of the *inferior oblique* of the *right* eye.

*Diplopia diminishes* when the gaze is *downward* and *towards the well side.*

The *face* is *directed upward* and *towards the diseased side*, and the *head inclines* over the shoulder of the *affected side.*

The *false projection* is *upward* and towards the *affected side.*

The *secondary deviation* of the sound eye is upward and towards the diseased side (*strabismus sursum vergens*, and *convergent*), with inclination of the vertical meridian towards the ill side.

Hence the corresponding diplopia is vertical and homonymous.

The image of the eye which does not fix (the healthy one) is the lower, and inclined towards the temple.

### SUPERIOR OBLIQUE.

In order thoroughly to understand the phenomena to which *paralysis of the superior oblique* gives rise, the pulley in the fossa trochlearis—that is to say, on the nasal side of the edge of the orbit, in front of and above the eye—must be regarded as its origin. From there, the muscle is directed obliquely *backward* and *outward*, and is inserted at the outer posterior and upper part of the eyeball, about as far to the rear of the equator as the insertion of the rectus superior is in front of it. (Fig. 10.)

Its muscular plane and its axis of rotation (*OO*) are virtually the same as those of the inferior oblique. The superior oblique directs the cornea *downward and outward*, and exerts upon the vertical meridian an *inclination towards the nose.*

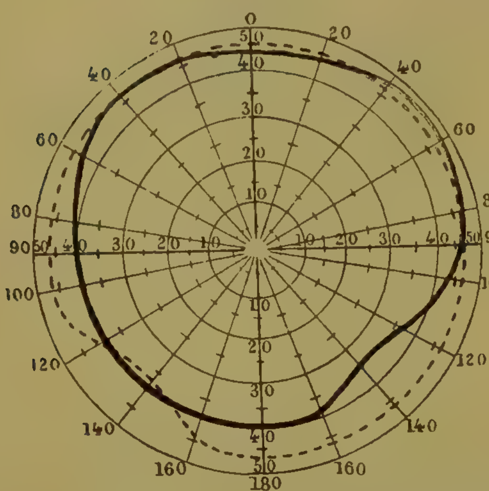
**Paresis of the Superior Oblique.**—The *excursion is restricted below and towards the affected side.* (Fig. 19.)

The eye is deviated upward and towards the healthy side (*strabismus*

*sursum vergens*, and slightly *convergent*), and the vertical meridian is inclined towards the temple.

The *false image* is below the real one and to the side of the ill eye (*vertical* and *homonymous diplopia*). It is, moreover, *inclined towards the healthy side*. (Fig. 20.)

FIG. 19.



Fixation-field of a *right* eye affected with *paresis* of the *superior oblique*. (Landolt, Arch. d'Opht., 1881, p. 606.)

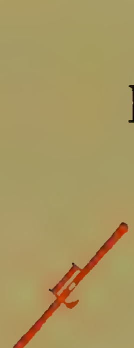
The *difference in height increases* when the patient looks *downward*, especially when the eye is directed *towards the healthy side*.

The *obliquity increases* when the gaze is *towards the diseased side*, for the reasons given when we were discussing paralysis of the inferior oblique.

Hence the *difference in height* and the *obliquity increase* and *decrease* in *inverse proportion*.

Vertical *diplopia* increases in proportion as the patient looks *downward* as well as towards the healthy side. *Obliquity increases* when the patient looks towards the *diseased* side.

FIG. 20*a*.




Position of the double images in *paralysis* of the *superior oblique* of the *left* eye.

FIG. 20*b*.


Position of the double images in *paralysis* of the *superior oblique* of the *right* eye.

The *false projection* is *downward* and *towards the diseased* side.

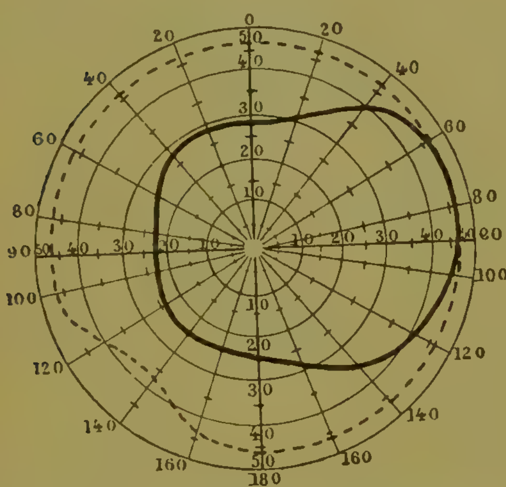
*Secondary Deviation.*—When the diseased eye is fixing, the healthy eye deviates downward and towards the affected side; its vertical meridian leans towards the temple. Hence the image belonging to it is above and on the same side (homonymous diplopia) and inclined to the affected side.

**Paresis of the Third Pair, or Common Motor Oculi.**—Among the muscles whose paralytic phenomena we have just been studying, there are only two—the first (*external rectus*) and the last (*superior oblique*)—that are innervated by a special cranial nerve; the former by the *sixth* pair (abducens) and the latter by the *fourth* pair (patheticus). The other muscles

—the *internal,* the *superior* and *inferior recti,* and the *inferior oblique*—are all animated by the *third* pair, the common motor oculi. That explains why isolated paralysis of the external rectus, as well as of the superior oblique, is not rare, while the other ocular muscles are almost always affected conjointly, at least to a certain degree.

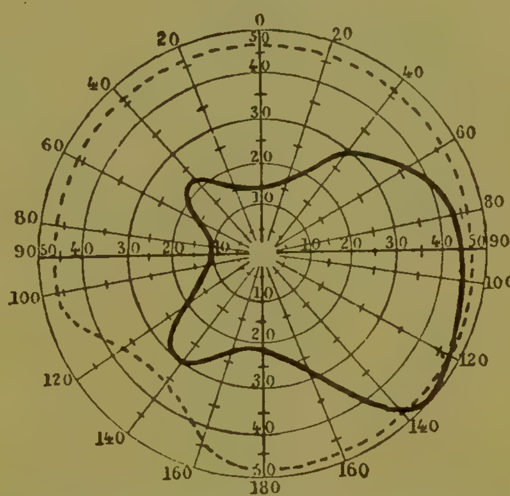
Therefore paresis of the common motor oculi will be characterized by a considerable limitation of the fixation-field. (Figs. 21 and 22.) Excursion *towards the healthy side, upward,* and *downward* will be noticeably *restricted.*

FIG. 21.



Fixation-field of a *right* eye having *paresis* of the *common motor oculi.*

FIG. 22.



Fixation-field of a *right* eye having *paresis* of the *common motor oculi* more advanced than that of Fig. 21.

The eye, whose external rectus and superior oblique act almost without antagonists, *deviates towards the diseased side* (*divergent strabismus*), and often somewhat *downward,* by virtue of the action of the superior oblique. The latter symptom is not always manifest, however, because of the abduction due to the external rectus, which interferes with the lowering action of the superior oblique.

The *nasal inclination* of the vertical meridian, produced by the superior oblique, is, on the contrary, rather marked, and becomes especially so when the patient looks downward, because then all the energy brought into play bears on this single remaining depressor.

The image belonging to the paralytic eye is on the opposite side (*crossed diplopia*), is generally somewhat higher and *inclined towards the temple,*—that is to say, towards the image belonging to the sound eye.

The *horizontal separation* of the two images *increases* when gazing *towards the healthy side.*

A *difference in height* is added to this and *increases* as soon as the patient looks *upward* or *downward.* In both cases, the false image seems to flee before the true one, so that it is the *higher* when the patient looks *upward,* and the *lower* when he looks *downward.*

The *obliquity* of the image *increases* especially when the patient looks *towards the affected side.* The horizontal diplopia *diminishes* at the same time.

The *head* is turned *towards the healthy* side and *upward*, especially when there is partial *ptosis*, and, moreover, is *inclined* over the shoulder of the *diseased* side.

The *false projection* is towards the *healthy* side.

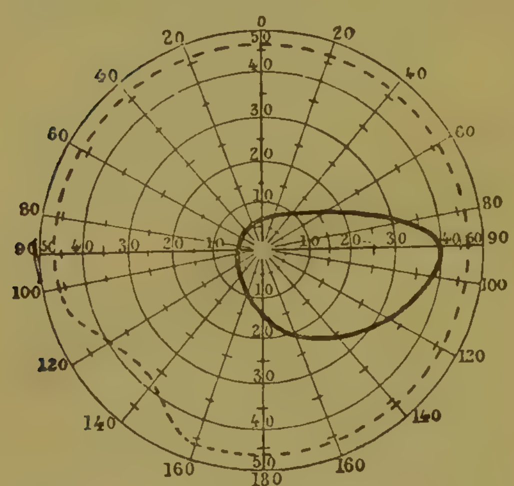
*Secondary deviation* consists especially in a strong divergence of the healthy eye.

Paralysis of the common motor oculi, moreover, most of the time produces that of the *levator of the upper lid*, as well as of the intrinsic muscles of the eye, the *sphincter pupillæ* and the *ciliary muscle*.

The phenomena to which it gives rise—the *ptosis*, *mydriasis*, and *paresis or paralysis of accommodation*—are most characteristic, and constitute, when they are combined with that of the *motor muscles*, an aggregate of symptoms concerning which one could scarcely err in diagnosis.

This is especially true when the paralysis of the third pair is complete. The upper lid is then entirely closed, and the frontal muscle, making strenuous but vain efforts to supply the action of the paralyzed levator, succeeds only in wrinkling the skin of the forehead, raising the eyebrow, and stretching the curtain lowered over the diseased eye.

Fig. 23.



Field of fixation of a *right* eye affected with *paresis* of the *oculo-motorius* and the *abducens*.

When this eye is uncovered, it is found to be totally divergent and almost immobile, with its pupil dilated to a certain degree.

It is evident that, in such a case, the sound eye could not, by secondary contractions, reproduce all the symptoms of the paralyzed eye and thus deceive the observer.

But we have called attention to the fact that, very often, the third pair is paralyzed for both eyes simultaneously: so that, in any case wherein there is complete paralysis of the common motor oculi of one side, there is occasion to seek also lesions in the domain of that of the other eye.

What is also interesting to note is that in almost all cases of this kind there exists a limitation of excursion also outward, in the direction in which the external rectus, freed from all opposition, ought to be able to turn the eye without obstacle. (Fig. 23.) This fact leads us to consider the

**Combined Paralyses of the Ocular Muscles.**—Although generally a single motor nerve, with the muscle or muscles which it innervates, is paralyzed, we sometimes see several muscles of the same eye or of both eyes simultaneously paralyzed.

We have already mentioned the limitation of the temporal excursions which almost always accompanies paralysis of the common motor oculi, and

which leads to a presumption of the simultaneous paresis of the abductor. (Fig. 23.)

We have also observed some cases of *double paralysis of the sixth pair.* (Figs. 24*a* and 24*b*.)

FIG. 24*a*.

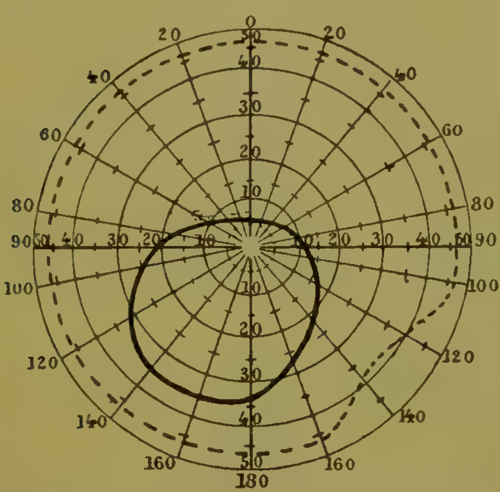
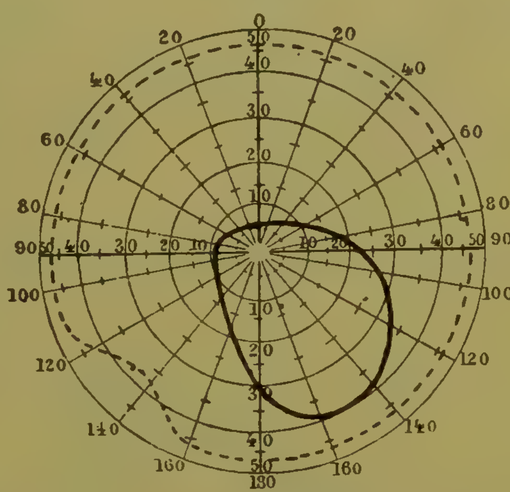


FIG. 24*b*.



Fixation-fields of both eyes of the same person affected by double paralysis of the third and of the sixth pair.

It happens, in the same way, that the *associated muscles,* as the *external rectus* of one and the *internal rectus* of the other eye, or *both levators* or *both depressors* of the two eyes, or the *two adductors* which preside over convergence, or the *two abductors* which preside over divergence, are paralyzed at the same time. The name ASSOCIATED PARALYSES has been given to those of the same type.

Finally, *all the motor muscles* of one eye—indeed, even those of both eyes—may be paralyzed. From this results a complete immobility of the eye, which is called *total ophthalmoplegia.*

If this concerns only one eye, nothing is stranger than to see the other move freely in all directions, while the affected eye remains stationary, as if fixed in its orbit. Usually ptosis preserves the patient from the intolerable diplopia which would result from the loss of the association of his eyes.

When the ophthalmoplegia is double, the patient is forced to make movements of his head take the place of those of his eyes. He will often be obliged even to use his hands to raise one of the upper lids which cover his pupils.

When one considers the smallness of the space whence arise the motor nerves of the eyes, the complexity of their cerebral paths, and the relatively narrow opening through which they enter the orbit, one is not astonished that the same morbid cause may affect several of them simultaneously.

The resulting symptoms are easy to deduce from what is above shown. The attentive observer will even recognize, without trouble, in each case, the implicated muscles.

If *paresis of the external rectus* is conjoint with that of the *common motor oculi,* the fixation-field will inform us of this fact, as in the cases

represented by Fig. 23 and Fig. 24. The crossed diplopia, characterizing paralysis of the third pair, may even, when the patient looks towards the affected side, be changed into homonymous diplopia, if the paralysis of the abducens is complete.

Paralysis of the *common motor oculi in both eyes,* as well as paresis of *both external recti,* is easy to recognize. The latter gives rise to *convergent strabismus* with *homonymous diplopia,* which *increases* towards *either side.*

The same is true of *paresis of both internal recti: divergence,* as well as *crossed diplopia, increases* towards the *right* as well as towards the *left.* Such a case is, however, a comparatively rare one. The weakness with which both internal recti seem struck at times, often concerns only their association in the movement of convergence. This will be discussed in the second part of the article.

The fixation-field will teach us, in such a case, concerning the power of the muscles in question. The nasal excursion of the eye is normal when it is a question of a functional disturbance of convergence, while it is limited in muscular paresis.

The *examination of the excursions* of the eyes will determine our diagnosis in all cases where diplopia is lacking.

### THE OPHTHALMOTROPE.

The influence of the different muscles upon the eyeball may be studied and demonstrated by means of an instrument we have had made, the *ophthalmotrope.* (Fig. 25.)

A schematic eye, consisting essentially of three bands of metal for its vertical and horizontal meridians and its equator, with a cornea attached, is so arranged as to be suspended in two stationary rings, one horizontal and the other vertical, by the three rotatory axes of the muscles.

In order to show the action of the *lateral recti,* we fasten the ends of the *vertical axis* by means of the two corresponding screws (*r*).

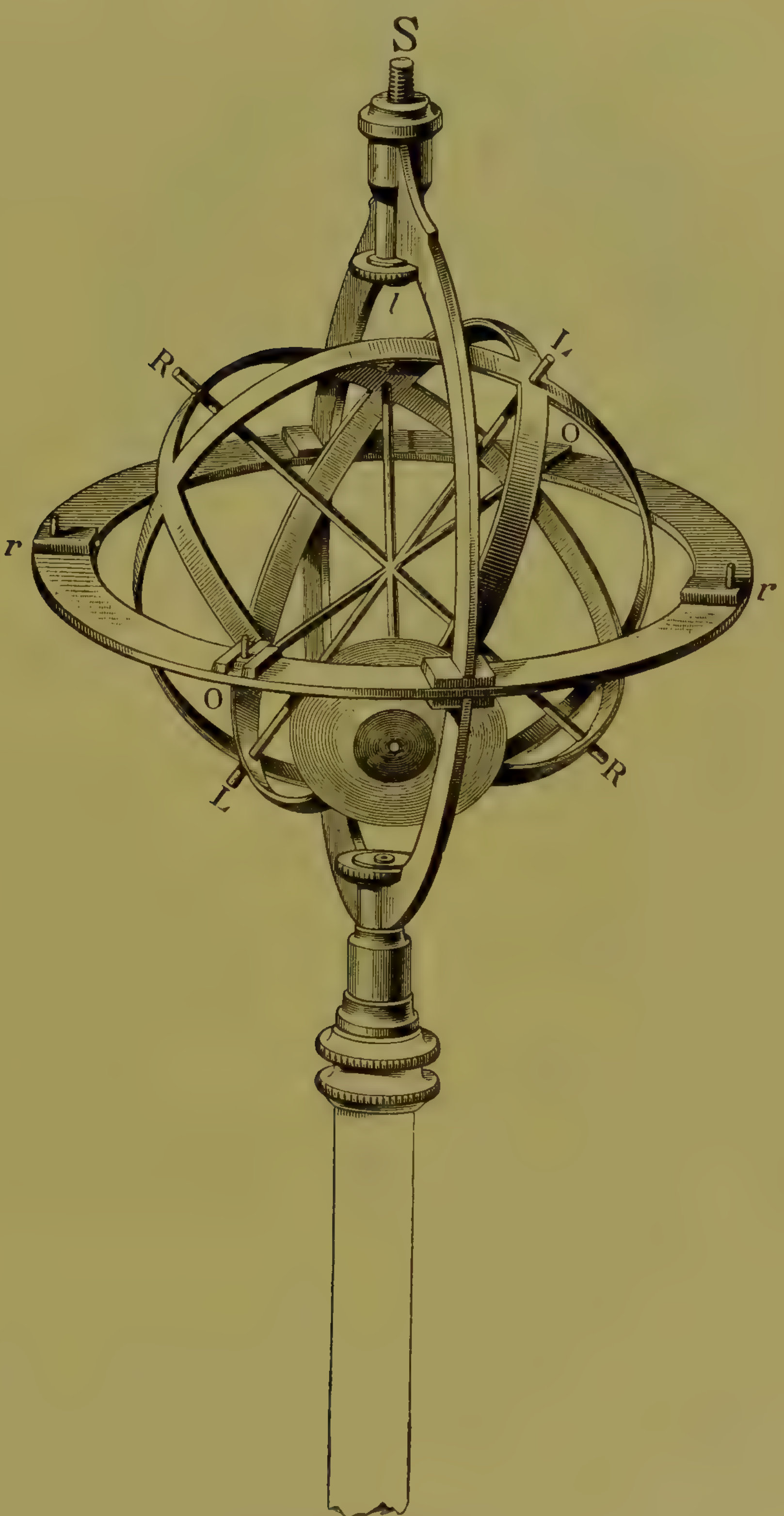
The axes of the *superior* and *inferior recti* (*RR*) and of the *oblique muscles* (*OO*) can be seized between similar screws connected with the *horizontal* ring. By fastening the screws, the eye is held in the position desired. In this way, even the most complex action of the ocular muscles may be studied with ease. Take, for instance, that of the obliques or of the superior and inferior recti. These muscles turn the cornea, so to speak, upon three axes: the *horizontal* (elevation and depression), the *vertical* (abduction and adduction), and the *antero-posterior* (inclination of the meridian).

Owing to the stationary rings of our instrument, which represent the immovable horizontal and vertical meridians, it is easy to see not only the position which these muscles give to the eye, but also the extent of each of the three rotations corresponding to a given degree of muscular contraction.

It is, further, interesting to compare the action of the vertical recti with

that of the oblique muscles. We may notice, for example, that, with the same degree of rotation, the inferior rectus lowers the cornea perceptibly

FIG. 25.




Landolt's ophthalmotrope.
Right eye under the influence of the superior oblique.

more than the superior oblique; but the inclination of the vertical meridian, as well as the lateral rotation, is more pronounced in the oblique than in

the rectus. Besides, these two movements are produced in the opposite direction for the two muscles.[1]

Our ophthalmotrope fulfils still another purpose. The posterior surface of the front half of the vertical meridian is painted in *red.*

This red line corresponds to the *false image* in the case of a *paralysis* of the muscle, the physiological action of which is shown by the ophthalmotrope.

In fact, as we have just shown, a muscle being paralyzed, the eye takes a position exactly the reverse of that which the same muscle, unparalyzed, would have given it. The retinal image is, on the other hand, projected in the sense opposite to the pathological position of the eye. The false image, therefore, corresponds in every way to the physiological action of the muscle.

For example, the *superior oblique* turns the eye *downward and outward,* and inclines the upper extremity of the vertical meridian *towards the nose.* In case of paralysis of this muscle, the eye is *deviated upward and inward,* and its vertical meridian is inclined *towards the temple.* The consequent *false image* is *inferior, temporal* (homonymous diplopia), and *inclined towards the nose.*

The *red meridian,* therefore, of the ophthalmotrope is identical with the *red lines* by which, in the preceding pages, we have indicated the image of the paralytic eye.

In fact, these lines are not approximately drawn, as has been the case hitherto, but are mathematically correct.

In Fig. 25, the ophthalmotrope represents the *right* eye under the influence of the *superior oblique* (cornea directed downward and outward; vertical meridian inclined towards the nose).

When unscrewed from the support and fixed by its opposite end ($S$), it represents the *left* eye under the influence of the opposite muscle, the inferior oblique, in our example.

The side of the eye (right or left), and the axes of the different pairs of muscles, are engraved upon the rings of the instrument.

In order to avoid confusion, different colors have been given to the different axes of the eye.

The action of the muscles upon the eyeball can be very simply represented in still another manner:

Take an ordinary rubber ball (Fig. 26); draw upon it the cornea, the vertical and the horizontal meridian. On the latter, mark the axis of the oblique muscles ($O$), 39° from the anterior pole (centre of the cornea), and on the opposite side ($R$) the axis of the vertical recti, 63° from the pole.

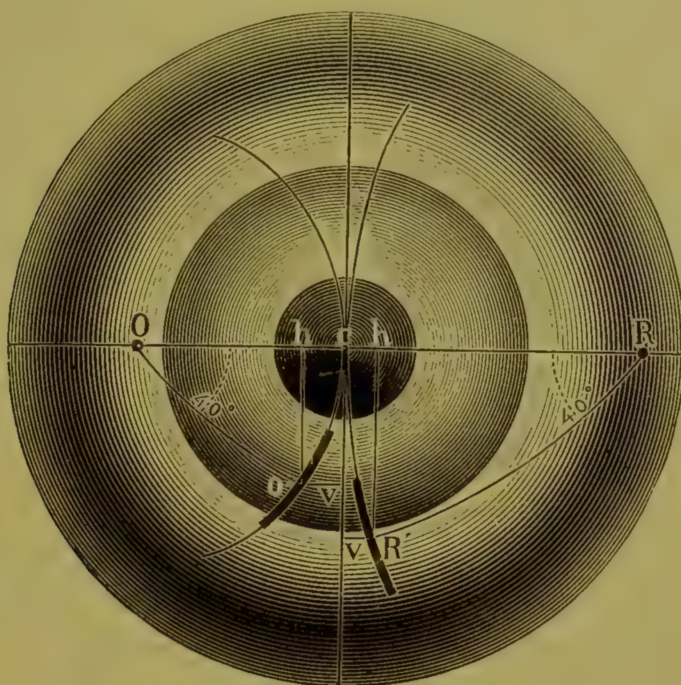
In order to demonstrate the action of, say, the *superior oblique,* we reason as follows: This muscle makes all points of the cornea describe parts of parallel circles around its axis.

---

[1] Compare Fig. 15 with Fig. 18, and Fig. 16 with Fig. 20.

Let us now take a compass and open it so that one of its points corresponds to this anterior extremity of the muscular axis, for instance $O$, the other to the centre of the cornea ($c$). Leaving the former fixed, we

FIG. 26.




Scheme of the right eye.

draw with the other, upon the ball, the circle of which the centre of the cornea describes a part ($cO'$, Fig. 26).

If we desire to know where this point is situated, for instance, after a rotation of 40°, we have only to trace, from the axis ($O$), a line (radius) forming an angle of 40° with the horizontal meridian (*below* it for the *superior*, *above* it for the *inferior* oblique).

The point where this line touches the circle corresponds to the centre of the cornea after the given rotation. Thus it is clearly seen how much the cornea has turned in the lateral as well as in the vertical sense.

As to the direction which the *vertical meridian* of the eye takes at the same time, it is evidently perpendicular to the radius $OO'$, and passes through the point which the latter has indicated to us as the centre of the cornea. This line is, in fact, no other than the horizontal meridian of the eye inclined by the muscular action. It is perpendicular to the vertical meridian.

Thus, in Fig. 26 the two black oblique tracks indicate the inclination of the vertical meridian produced, on the right eye, by the superior oblique ($O'$) and the inferior rectus ($R'$), after a rotation of 40° round their axes.

On projecting upward vertical lines from the points $O'$ and $R'$ to the *horizontal* meridian ($O'h$ and $R'h$), we obtain the amount of *depression* of the globe produced by the muscles in question.

The perpendiculars $O'v$ and $R'v$, which are drawn from the points $O'$ and $R'$ to the *vertical* meridian, correspond to the degree of *abduction* ($O'v$), caused by the oblique, and of *adduction* ($R'v$), caused by the rectus muscle.

## CLINICAL DIAGNOSIS OF PARALYSIS OF THE MOTOR MUSCLES OF THE EYE.

What we have said in the foregoing sections concerning the symptoms of paralysis of the various muscles of the eye should, it seems to us, be sufficient to help the reader to pass in this matter a pretty good examination. But it is not an examination of this kind to which our patients subject us; they do not come with the label of a muscular paralysis, asking us what were its symptoms; on the contrary, they present to us the symptoms from which we are to deduce the paretic or paralyzed muscle or muscles.

Before going farther, we therefore wish to show the reader the method which we follow customarily, in order to get our bearings in the multitude of complaints to which a muscular paralysis may give rise, to seize, among them, the characteristic symptoms, and to reach the desired diagnosis most easily and in the surest possible way.

When a paralysis is complete, it produces a deviation and a limitation of the excursions of the eye of such kinds that the simplest examination suffices for the discovery of the muscle or muscles which are out of use.

But paralyses of this sort are not very common; far more frequently we meet with paresis. He who should hope to recognize such at the first glance, by means of the strabismus which they ought to provoke, would expose himself not only to bitter delusions, but also to the gravest misapprehensions.

We shall see farther on, when discussing strabometry, that the eyes sometimes appear to diverge or converge by virtue of the direction of their pupillary axes, while their visual lines, which alone determine the direction of the gaze, may be normally directed, or be deviated in the same sense as the pupils, or even in the inverse sense.

Still more deceptive are the differences in height of the eyes. If one will direct his attention carefully to this matter, it will be found that very often one eye is sensibly higher than the other, in persons who enjoy a perfect field of binocular fixation. Of course in such a case it is not a question of the vicious direction of the lines of sight, but of the irregularity of location of the eyes in their orbits, an irregularity or asymmetry which, happily, is counterbalanced by the wonderful action of the motor apparatus.

Thus, I discovered one day accidentally in one of my friends, a general officer, that one of his eyes was at least five millimetres higher than the other. This military gentleman never has perceived, however, any anomaly in his vision, and the attentive examination which he permitted me to make proved that both his eyes were emmetropic and enjoyed normal acuteness of vision, and that their movements harmonized perfectly in every direction.

Moreover, I have had the opportunity of showing to my pupils a patient affected with vertical diplopia. The left eye was visibly lower than the

right. Hence one might well presume the paresis of a levator muscle of that eye, or that of a depressor of its congener. But the examination demonstrated that it was a question, on the contrary, of the paresis of the superior oblique,—that is to say, of a *depressor* of the same left eye. Though it was below the level of the right eye, the left eye was unquestionably directed towards a higher point than that towards which tended the line of sight of the other eye.

It is thus evident to how many errors we should expose ourselves in basing upon appearance only our judgment concerning the direction of the eyes. In fact, the *subjective symptoms* of paresis of an ocular muscle are much more significant than the exterior aspect of the eyes.

But here, again, we must not hope that the patient will inform us, at the outset, of the diplopia characteristic of the paralysis of each ocular muscle. It even happens that a patient may complain of double sight, when his muscles are performing their functions quite normally. In such a case we are concerned with *monocular diplopia*, which may have various causes entirely independent of the motor apparatus.

On the other hand, persons who are victims of muscular paralysis are often unaware of their diplopia. They say that their vision is *dull*, but not that it is *double*. The most frequent symptoms of muscular paralysis are rather a feeling of extreme annoyance, of pain in the head, of difficulty in getting about, vertigo, or an indefinite malaise, which may go to the extent of vomiting, etc.

Very often, also, the gait of such patients betrays their infirmity, and he who follows our advice to observe the patient at a distance and in his entire behavior will discover a multitude of most precious signs for the diagnosis, which signs will escape him who fixes his attention, from the outset, upon the eye which appears to him to be affected.[1]

Thus, persons afflicted with muscular paralysis are generally distinguishable by an *uncertain gait*. They sometimes seem dazzled, or as if they were afraid of jostling against objects about them. They rarely walk with the head straight, and, if they are seated in front of us, they almost always look at us more or less obliquely; often, too, they close one eye in order to see well, and only then seem entirely calm and satisfied.

Insist, then, upon the patient opening both his eyes, and get him to go to the other end of the room and return rapidly towards you. Observe if he does not stagger as if the worse for liquor; if one side of the body is not in advance of the other as he walks; if he does not look at you over one shoulder; if he does not tip the head backward or forward, or turn it towards the right or the left. Such are the symptoms which not only cause you to suspect the presence of a muscular paresis, but often even put you in the way of finding the muscle affected.

The patient who turns his *face* towards the *left*, in order to look at you,

[1] Landolt, Manual of the Examination of the Eyes, Philadelphia, 1879, p. 21.

is probably affected in his *left external* or his *right internal* rectus; while one of the opposite muscles would be likely to be defective in the case of him who looks at you with his face turned towards the right.—A head inclined *backward* (nose in the air) gives rise to the presumption of a paresis of a *levator;* a *bowed* head (forehead most advanced) is likely to indicate a paresis of a depressor.—A head *leaning to one shoulder* ought to awaken the suspicion of paralysis of one of the muscles which turn the eye around the antero-posterior axis,—in other words, *incline the vertical meridian.*

After having thus informed yourself as to the position of the body and head, and what seems to help the patient's vision most, then do the opposite. Place the patient in front of you, his head quite straight, and make him open both his eyes. Without attaching too much importance to an apparent deviation under such circumstances, it is nevertheless permissible to take cognizance of it. But a truly pathological deviation will be manifest only when, making him fix some object, we *cover and uncover the eyes alternately.* Thus it is that we may assure ourselves whether both eyes are simultaneously directed towards the same point, or whether, when excluded from vision, they deviate, and in which sense.

Remark especially if the deviation is the *same for both eyes,* or if it is *more pronounced in one than in the other.* This phenomenon is, as we have previously shown, pathognomonic of a muscular paralysis, and indicates, at the same time, which eye is affected. The paralyzed eye is that which deviates *less.*

As fixation-object the index finger, held in the median line before the patient, is customarily chosen. This simple method is not altogether bad, but it could be better. The finger, held near the patient's nose, obliges him to converge. Now, if he is the victim of convergent strabismus, the direction which we force him to give to his eyes masks for us, at least in part, his real infirmity, which would become much more apparent if he were looking at a distant object, because such distant vision calls for parallelism of the visual lines.

Hence we advise that the *distance* of the fixation-object be *varied;* that it be at first a near object, then a distant object.—But what is especially important is that the *direction* of the object fixed be *varied.* The finger should be moved to the left, to the right, up, down, or, again, the direction of the patient's gaze is to be varied, as we shall see, by changing the direction of his head.

This summary inspection having been carefully made, have recourse to a symptom which is much more important and much more apt to fix the diagnosis,—that is to say, to the *diplopia.* It will never be lacking, provided that binocular vision has existed before the malady, and that one knows how to provoke it.

In order to produce diplopia, we use as fixation-object a candle flame which stands out boldly upon a *dark* background. We place it at a distance of at least two metres, in order that the separation of the two images may

be notable enough for a deviation of even a few degrees. We cover one of the patient's eyes—generally the one which has the better visual acuity—with a colored glass. It is best to choose a *red* glass of not too deep a shade. This changes the color of the flame sufficiently to distinguish it from that seen by the other eye, but does not deprive it of so much luminous intensity that the patient may fail to perceive it.

Then the patient opens both eyes and holds his head erect and motionless, and we ask him if he sees one light or two lights? If he admits only one, we ask him the color of it, and we cover, for an instant, the eye with which he sees it, in order to attract his attention to the image received by the other eye. In this way, one generally succeeds readily enough in rendering the diplopia manifest. If not, the direction of the gaze is to be varied, for it might happen that the defect in motility would be weak enough not to interfere with binocular vision while the patient is looking straight ahead, but only for certain directions of the eyes. We know, indeed, that diplopia manifests itself especially in the direction towards which the enfeebled muscle turns the eye.

On the other hand, it has been observed that pathological *convergence* has a tendency to *increase* when the gaze is *downward*, while *divergent* strabismus is especially shown when the patient looks *upward*. This peculiarity is probably due to the fact that it is generally in order to observe near objects, which necessitates an effort of convergence, that we look downward; while we direct the gaze upward to look at infinite distance, to relax our adductor muscles and abandon the eyes to the abductors.

In order to change the direction of the gaze, it is customary to displace the fixation-object. A simpler and better method is to leave the flame where it is, and to *change the direction of the patient's head.* Thus, in order to make him look upward, we direct his face downward; in order to make him look downward, we direct his face upward; in order to make him look towards the right or the left, we turn his face to the left or to the right, and the same for intermediate directions. In this way, not only do we avoid promenades with the lighted candle, not only do we always control the position of the patient's head, but we do away with a cause of error which has led more than one master astray.

It happens, indeed, at times, that the patient, instead of seeing the images separated horizontally or vertically, declares that one of them comes towards him. He then loses all notion as to the relative distance of the two images. This fact is capable of easy explanation, as follows:

Let Fig. 27, $ABCD$, be our consultation-room, $P$ the patient's head, $F$ the candle flame, placed against the wall opposite him.

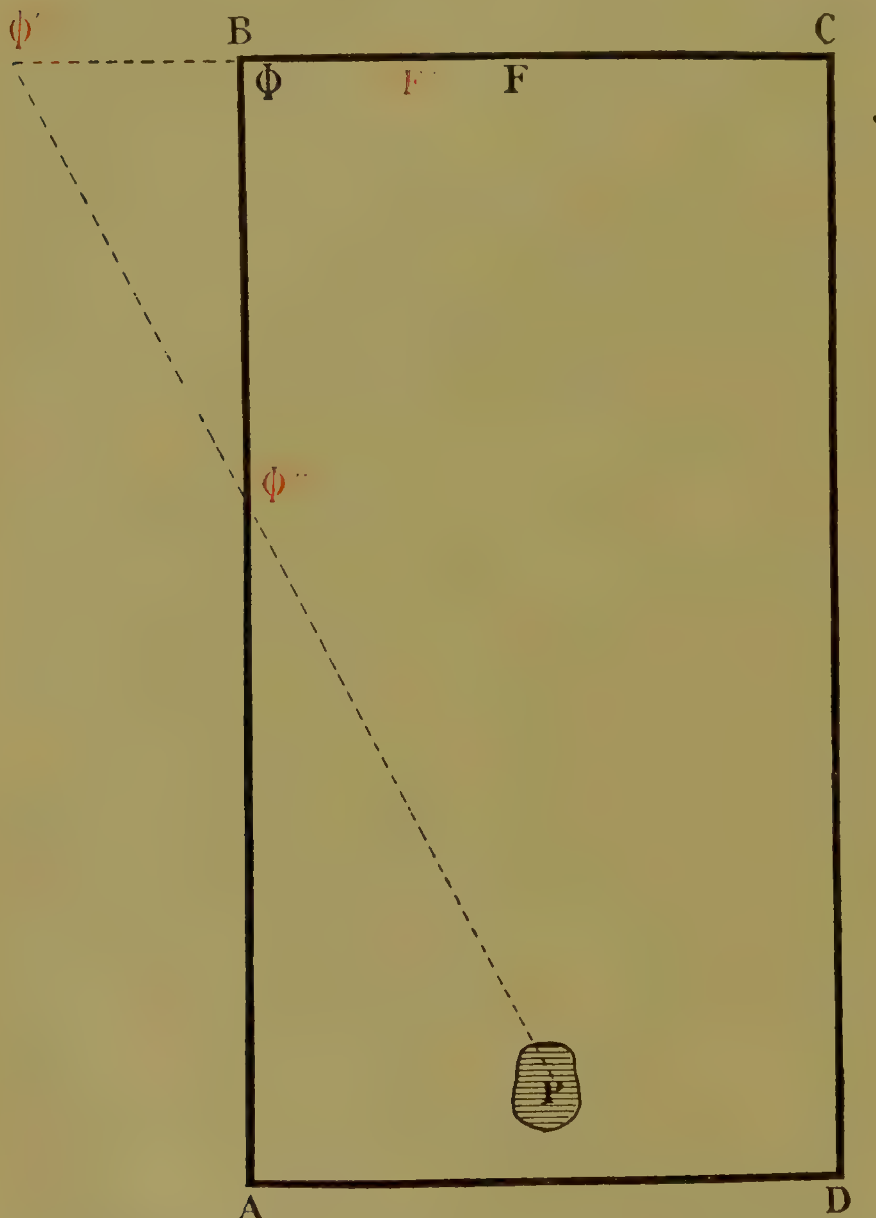
If he has *paralysis of the left external rectus*, he will see this flame at $F$ with his right eye, and a second flame at $F'$ with the left eye.

If now we carry the flame from $F$ to $\Phi$, the separation of the two flames increases, and the patient ought to see one flame at $\Phi$ and the other at $\Phi'$, as if it were behind the wall $AB$ of the room. It is true that the image of

this wall is also doubled, but its image cannot be as vividly perceived by the ill eye as can the image of the flame.

However this may be, the patient sometimes supposes the flame seen with the diseased eye nearer to him, on the wall $AB$, say at $\Phi''$, and if we ask him, "Is the red light nearer or farther?" (meaning relatively to the

FIG. 27.




naturally colored light), he will answer, "It is nearer" (he referring to himself).

Even if we succeed in rendering our question comprehensible to the patient, it is sometimes very difficult to establish whether the separation between the two images is increased or diminished, inasmuch as the patient is often deceived, in such a case, and even declares that the two images are nearer each other, when they notoriously are farther apart.

The same thing occurs for the vertical diplopia when the object of fixation is raised or lowered.

Let us, for instance, turn Fig. 27 ninety degrees to the left. Then $AB$

will represent the floor and *CD* the ceiling of the consultation-room. Instead of supposing that one of the flames is lowered into the cellar at $\Phi'$, the patient, subject to paralysis of a depressor, supposes that it is advanced towards him, on the floor, to $\Phi''$.

We put an end to all these embarrassments in changing the *direction of the gaze by altering that of the patient's head.* In order to cause him to look towards the left, we turn his head (that is to say, his face) towards the right. To make him look to the right, we direct the face towards the left. To turn his gaze upward, we incline the face downward, and to lower the gaze, we direct the face upward. In this way, the plane of projection of the double images always remains the same. It is the wall on the surface of which is placed the flame.

Diplopia having been obtained, we first take into account in what sense it is produced, whether in the horizontal, the vertical, or in both directions at once; if it be homonymous or crossed, vertical or intermediate.[1]

After having informed ourselves concerning the diplopia, while the patient is looking straight ahead, we change the direction of the gaze (by means of movements of the head), asking him if the two images separate or if they approach each other.

We thus find, at once, not only which is the affected eye, but also the direction of the action of the implicated muscle,—indeed, even the muscle itself, when it is a question of one of the lateral recti.

In fact, *the affected eye is that in the direction of the image of which the diplopia increases, and the paretic muscle is that which turns the eye in the sense of the false image.*

Example:

The *left* eye being covered with a *red* glass, the patient announces *homonymous* diplopia; the left eye's image is to the left, that of the right eye to the right. We direct the patient's gaze towards the *left* by turning his head towards the right. If the double images get farther apart, it is the *left* eye which is the affected one, because it is in the direction of its image that the diplopia increases, and it is its *external rectus* which is affected, for it is that muscle which directs the eye towards the false image,—that is to say, towards the left.

If there be *crossed* diplopia, the (red) image belonging to the *left* eye is at the *right*, that belonging to the *right* eye is at the *left*. If then the diplopia increases when the gaze is directed towards the *left*, it is the *right* eye

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[1] It is to be noticed that the patient sometimes speaks of a certain difference in the level of the double images as accompanying a horizontal diplopia. This is often due to an unconscious inclination of the head, or to the fact that an eye which is subject to a very pronounced convergent strabismus usually deviates slightly upward, while high degrees of divergence are readily combined with a certain lowering of the eye. One can then bring the two images back to the same level by rectifying the position of the patient's head.

There is reason to suspect a complication with the paresis of a muscle acting vertically only when the difference in height does not admit of so simple correction, and especially when it is more marked than the horizontal diplopia.

which is the affected one, inasmuch as, this time, it is its image which is at the left. Moreover, we shall know that we are concerned with paresis of the *internal rectus*, because it is that muscle which turns the right eye towards the left.

Let us now take the case of a *vertical diplopia;* the two images are *superposed.* This case is a rather complicated one, inasmuch as there are no less than eight muscles whose paralysis may provoke this phenomenon. But with the aid of our law we easily regain our bearings.

The *left* eye being still covered by the *red* glass, we ask the patient whether the red flame is above or below the other.

Suppose the patient answers that it is *below.* If now, in *lowering* the gaze, the images separate still farther, we shall know that it is a question of the paralysis of a *depressor* of the left eye. If they approach each other, it is one of the levators of the right eye which is paretic. Indeed, if, on the contrary, we make the patient look upward in order to see the fixation-object, the diplopia is increased. It increases in the direction of the higher image,—that is to say, that of the *right* eye.

But suppose we are concerned with *paresis of a depressor of the left eye.* It remains for us to determine whether it be the *inferior rectus* or the *superior oblique* which is paretic.

We bear in mind that the former is, at the same time, an *adductor*, and that, in consequence, its paralysis must bring about, besides the raising of the eye, a *divergent strabismus* with *crossed diplopia*, while paralysis of the *superior oblique* has *convergence* and *homonymous diplopia* as its accompaniments.

We therefore ask the patient if the two flames that he sees are exactly in the same vertical, or if one of them is a little to the right or left of the other. If he answers this question clearly, our diagnosis is complete. But very often this horizontal deviation is not well marked, or the patient is not sufficiently aware of it; his answers are undecided and ill adapted to inform us concerning the exact position of the deviated eye.

We might then think of having recourse to a third rotation that an eye whose depressor is paralyzed undergoes, the rotation around the antero-posterior axis, the *inclination of the vertical meridian*, which is *nasal* for the *rectus* muscle, *temporal* for the *oblique.* But if the patient be not aware of the horizontal separation of the two images he will habitually recognize still less the obliquity of one of them.

Hence one would be much embarrassed to make a diagnosis in such a case, if there were not some means for rendering the character of the diplopia more clear and apparent. This means consists again in the variation of the direction of the gaze.

Direct the patient's face to the left, so that he is obliged to *turn his eyes towards the right* in order to see the object. If, in this case, the *difference in height* of the double images *diminishes*, while a certain degree of *crossed diplopia*, and, especially, an *inclination* of the lower image towards the

*temple*, is *manifested*, we have to do with paresis of the *inferior rectus*. (See page 25.)—The inverse will occur, when the gaze is towards the left, the direction of the face towards the right in our example.

On the contrary, *increase of the vertical separation*, when the eyes are directed to the *right* (in our example), *diminution* of the *difference in height*, when the gaze is to the *left*, with apparition of increasing *nasal obliquity*, characterize paresis of the *superior oblique*.

There is a very easy means of recalling instantaneously the variation which the diplopia caused by paralysis of a depressor or levator undergoes in the different directions of the gaze. One needs only to remember the *direction of the muscles*, and to say that *the more the gaze* (the axis of the eye) *approaches this direction*, the more the action of the muscle in the *vertical* (lowering or raising) becomes *manifest;* and that, on the contrary, the more the eye *deviates from the direction of the muscle*, the more the latter becomes a rotator around the antero-posterior axis, the more *the inclination of the meridian* becomes *manifest*.

The *recti muscles* (superior and inferior) are directed towards the *temple*. Consequently an *increase* in the *vertical diplopia*, when the patient looks towards the *temple* of the affected side, indicates the paralysis of a rectus muscle. In order to make the diagnosis still more certain, the patient will be required to look in the opposite direction, and the *obliquity* of one of the images will be manifested, at the same time that the difference in height will diminish.

The *oblique muscles* may be considered as being directed towards the *nose*. Hence, if the *vertical diplopia is increased* when the patient directs the paralyzed eye towards the *nose*, it is one of the oblique muscles which is affected.—Inversely, the double images come closer to each other, and the *obliquity* of one of them *increases*, when the gaze is towards the *temple* of the affected side.

The patient's appreciation of the obliquity of one of these images may sometimes give rise to an error. On looking to the left, he will, perhaps, be clearly aware of their mutual approach and of the obliquity of one of them. We are then much gratified to state the perfect concordance of the ascertained symptoms with what we have foreseen; but, when we say to the patient, "Of course the lower flame leans towards the right?" "Not at all," he objects; "the lower one is perfectly upright; it is the upper one, which leans towards the left."—Now, the upper image belongs to the right eye, which, up to this time, we have regarded as healthy. Have we deceived ourselves as to the eye, or is there some complication on the part of the right eye,—some paresis, some contraction? No; the appreciation of the inclination of a line depends on what one regards as the vertical; in our example, the two flames lean towards each other. If the patient bases his observation upon that which belongs to the right eye, in order to appreciate the inclination of the other, he will say, as we had hoped, that the lower flame leans towards the right. If, on the contrary, he takes as

standard the lower light, it is the other one which is not upright, but whose upper extremity leans towards the left.

It is as if, in a case of paralysis of the left abductor, this eye being covered with a red glass, we ask him, "The red flame is to the left, the yellow to the right, isn't it?" and he answers, "No, the yellow is in the middle and the red is to the left;" or, again, "The red is in the middle and the yellow to the right." It is simply a question of personal appreciation.

The examples cited will suffice to enable the reader to apply efficaciously the theories brought out in the preceding paragraphs, and to succeed, even with patients of defective intelligence, in reaching a precise diagnosis of every muscular paresis or paralysis.

The attentive clinician will find then, to his great satisfaction, that the so-called complicated, obscure cases that do not fall in with the laws of physiology are of very rare occurrence. For our part, we have never yet met an "obscure" case in which some lack of attention on our part or on that of the patient was not infinitely more probable than any defect in the laws of physiology, whence those of pathology are necessarily and logically derived.

## STRABOMETRY.

In order to become fully acquainted with a case of strabismus, it is necessary to determine the *degree* of it; to add to its qualitative, so to say, its quantitative analysis.

*Strabometry* is the name we give to this detailed analysis of an ocular deviation. It comprises the determination of the degree of the deviation —that is to say, of the *angle of strabismus*—and the examination of the monocular and binocular excursions, of the *monocular fields of fixation,* as well as of the *binocular field of fixation.*

### DETERMINATION OF THE ANGLE OF STRABISMUS.

We have given to the angle of strabismus the following definition: *the angle of the strabismus is the angle comprised between the direction which the line of sight of an eye has and that which it ought to have.*[1]

The simplest method for solving this problem *objectively* is the following:[2]

The deviated eye is placed at the centre of a semicircle, movable about its summit, which summit is at the same time the zero-point of a division which goes, on either side, to 90°. Our perimeter is well adapted to this use; its arc (*PoP*, Fig. 28) is placed in the plane of the deviation of the eye, horizontal for lateral strabismus, vertical for vertical strabismus, obliquely for intermediate strabismus, unless one prefers to divide the latter form of squint into two components, the one horizontal and the other vertical.

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[1] Landolt, Graefe und Saemisch, Handbuch der gesammten Augenheilkunde, iii. S. 325.

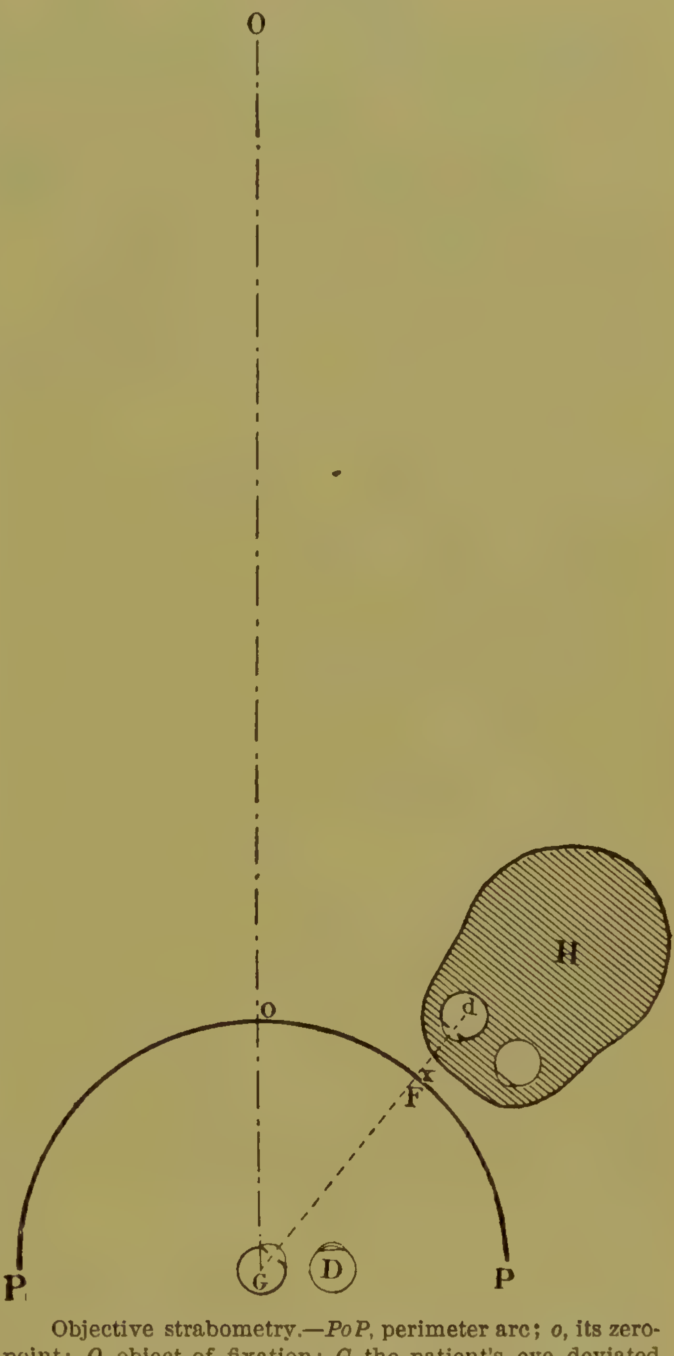
[2] It is based upon a principle pointed out by M. Javal.

The patient is made to fix the point $O$ (Fig. 28) situated in the prolongation of the radius of the arc, which passes through its summit $o$. In order to avoid the error which might result from convergence, we select, for an object of fixation, one as far distant as possible, at least five metres away.

The sound eye ($D$) alone will be directed towards the object. If the affected eye ($G$) were equally so, its line of sight would necessarily pass through the point $o$ of the arc. Hence $GoO$ is the direction which the eye *ought to have.* But as it is, in reality, deviated, its line of sight passes through some other point of the arc. The corresponding division $x$ will give us the angle of the strabismus, $oGx$. How is this point $x$ to be determined?

If the line of sight passed through a known and visible point of the globe, nothing would be easier than to ascertain, *objectively*, its direction. But such is not the case; we can only presume that it does not pass far from the *centre of the pupil.*

FIG. 28.




Objective strabometry.—$PoP$, perimeter arc; $o$, its zero-point; $O$, object of fixation; $G$, the patient's eye deviated towards $F$; $d$, the observer's eye.

Hence, for an approximate appreciation, we simply seek the point of the perimetric arc towards which is directed the ray of the cornea which passes through the pupillary centre.

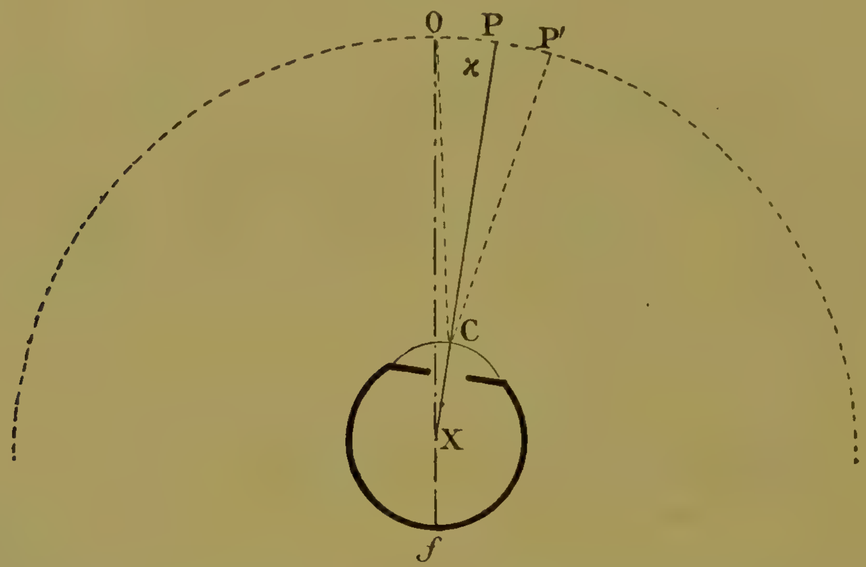
For that purpose, we move, along the inner surface of the arc, a small lighted candle, $F$ (Fig. 28), whose movement we follow with one of our eyes, $d$ (the other being closed), in such a way that our eye and the flame are always on the same radius of the perimeter. The point of the arc where the candle must be placed, in order that its reflex shall appear just at the centre of the pupil of the observed eye, is then that towards which the radius of the cornea which passes through the pupillary centre is

directed. For simplicity's sake, we will call this the *pupillary* axis, and the degree on the arc designates what I call the *apparent strabismus*.[1]

One may be content with the degree of the apparent strabismus when it is not a question of great precision, or in the case of amaurosis of the deviated eye, for when there is no vision there is no visual line.

But when one wishes to know the *real strabismus*, the degree of the deviation of the visual line, then it is necessary also to determine the angle separating the pupillary axis from the visual line. I have given to this angle the sign $x$ and the name *Kappa*.[2]

FIG. 29.




The angle Kappa may be determined, with sufficient precision for practice, in the following way: The affected eye being placed at the centre of the perimeter, we cover the sound eye, and have the patient fix the flame at the summit of the arc. In this case the visual line is necessarily directed towards this zero-point, *O* (Fig. 29). If the visual line coincided with the pupillary axis, the reflex of the flame would appear to be at the centre of the pupil, when we look at the observed eye from the summit of the arc. This may occur, but it is not the rule; in general, the pupillary axis passes to the outside or to the inside of the visual line.

To find at once the direction and the degree of the angle Kappa, we leave the flame immobile at the zero-point, and we move our eye along the arc until we have found the point at which it is necessary to view the observed eye in order that the reflex of the flame may appear at the centre of the pupil. The corresponding degree on the arc then represents *twice* the angle Kappa.

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[1] It has been sought to base such researches upon the centre of the cornea. But, aside from the fact that the centre of the cornea is more difficult to find than that of the pupil, it is in no wise the cornea, but the pupil, which determines, for the observer, the direction of the gaze of an eye, and, consequently, the apparent strabismus.

[2] Landolt, Traité complet d'Ophthalmologie, par De Wecker et Landolt, iii., p. 815, 1887.

The explanation of this is very simple: Let $fO$ (Fig. 29) be the visual line of the eye, $XP$ the pupillary axis. $OXP$ will be the angle Kappa. If the flame remain at $O$, I am obliged to move my eye to $P'$ in order to see, at the centre of the pupil, its reflex formed by the portion $C$ of the cornea. Now, the angle $OCP'$ represents the sum of the angle of incidence $OCP$ and the angle of reflection $PCP'$, which are equal. The angle of incidence $OCP$, being nearly equal to the angle Kappa, may be regarded as the double of the angle Kappa.

It might seem that it would be simpler to have the patient fix the zero-point of the arc, and to carry the candle along the arc, in order to find, at $P$, directly, the angle Kappa. But this angle is usually so small that we are glad to possess, for its rapid determination, a method which gives a precision double that of this direct method.

Usually the pupillary axis passes *without*—that is to say, at the *temporal* side of—the visual line (*positive* angle Kappa, $+\varkappa$). This condition produces for the spectator (who has for his guide only the pupils which he sees, and not the visual lines which he does not see) the appearance of a *divergent strabismus*, even when the eyes of the person observed are normally directed. Thus, in case of *positive* angle Kappa, a real divergence appears exaggerated, while a convergent strabismus is apparently diminished—indeed, often entirely concealed.

To find the real angle of strabismus it is, therefore, necessary to *subtract* the *positive* angle Kappa from the apparent *divergent* strabismus, and to *add* it to the apparent *convergent* strabismus.

The angle Kappa takes the *negative* sign ($-\varkappa$) when the pupillary axis passes to the *inside*—to the *nasal* side—of the visual line. In this case there is an *apparent convergence* during the normal direction of the gaze. Hence the *negative* angle Kappa ought to be *added* to the angle of the apparent *divergent* strabismus, inasmuch as the lines of sight diverge even when the eyes have the appearance of being parallel. It ought to be *subtracted* from the angle of the apparent *convergent* strabismus, which it causes to appear unduly great.

## SUBJECTIVE STRABOMETRY.

The *diplopia* which we have met as one of the principal symptoms of paralytic strabismus permits us to determine the angle of strabismus much more easily and exactly than by the preceding method.

The plan of *subjective strabometry* that we have pointed out[1] is based on the fact that the distance between the double images is the *tangent* of the *angle of strabismus.*

We need only refer to Fig. 2, in which $O$ is the fixation-object, $\Omega$ the point towards which the deviated eye is directed, and $Om\Omega$ the angle of strabismus.

[1] Landolt, Annales d'oculistique, Juillet, 1875. See also Hirschberg, in Archives of Ophthalmology, iv., September, 1875.

In order to find the point $O'$ where the deviated eye supposes the object $O$ to be, we have been obliged to bring this eye back to its normal direction, to make it execute a rotation equal to its angle of strabismus in the inverse sense. Hence the angle $OmO'$ is equal to the angle of deviation $Om\Omega$; the distance separating the two images, $O$, seen by the healthy eye, and $O'$, seen by the affected eye, is, therefore, the tangent as well of the angle $OmO'$ as of the angle of strabismus.

It suffices to measure this distance as well as that which separates the eye from the object of fixation, to obtain the angle of deviation,—in other words, the angle of strabismus,—by means of a simple trigonometric formula.

To avoid any calculation, we have divided into tangents of multiples of 5° one of the walls of our consultation-room. This division is, of course, applicable to only one radius,—that is to say, to a given distance. It is therefore necessary that the person examined shall always occupy the same position relatively to the wall. For that purpose, we have marked, by four nails driven into the floor, the location of the chair occupied by the patient. On the wall opposite to him, on a level with his eyes, is the zero-point of our division, which goes from there to the right, to the left, upward and downward, and in two intermediate directions. The patient is, so to say, within a sphere whose centre coincides with his eyes, while the points where the radii, going thence, reach the wall, are designated by figures.

If we place, on the wall, a candle flame at the zero-point of the division, the patient needs only to point out the place at which he sees the second image, and we read there directly the angle of his strabismus. We may similarly appreciate the increase and the diminution of this angle in the different directions of the gaze.

For a distance of *three metres*, the tangents—that is to say, the divisions of the wall—are the following:

Starting from the zero-point,

| | | |
|---|---|---|
| 5° = 26 cm. | 20° = 109 cm. | 35° = 210 cm. |
| 10° = 53 cm. | 25° = 140 cm. | 40° = 251 cm. |
| 15° = 80 cm. | 30° = 173 cm. | 45° = 300 cm. |

The upper part of the vertical meridian bears the same division. It is convenient, however, to continue it further, and to mark

| | |
|---|---|
| 50° = 358 cm. | 55° = 428 cm. |

The lower part is divided in the same way as far as to the floor. The latter will be found eleven centimetres below the number 20° of the division.

From this point (120 cm. from the zero-point) the degrees of the division present the following distances:

| | | |
|---|---|---|
| 25° = 43 cm. | 40° = 157 cm. | 55° = 216 cm. |
| 30° = 92 cm. | 45° = 180 cm. | 60° = 231 cm. |
| 35° = 129 cm. | 50° = 200 cm. | |

For a radius of *two hundred and twenty-five centimetres* (the distance from the patient to the wall) the divisions of the horizontal meridian correspond to the following numbers:

Starting from the zero-point,

| | | |
|---|---|---|
| 5° = 19.6 cm. | 20° = 82 cm. | 35° = 158 cm. |
| 10° = 39.6 cm. | 25° = 105 cm. | 40° = 189 cm. |
| 15° = 60 cm. | 30° = 130 cm. | 45° = 225 cm. |

For the ascending part of the vertical meridian,

| | |
|---|---|
| 50° = 274 cm. | 55° = 321 cm. |

The descending part touches the floor at one hundred and twenty centimetres,—that is to say, between 25° and 30°.

From this point (120 cm. from the zero-point) the different degrees correspond to the following lengths:

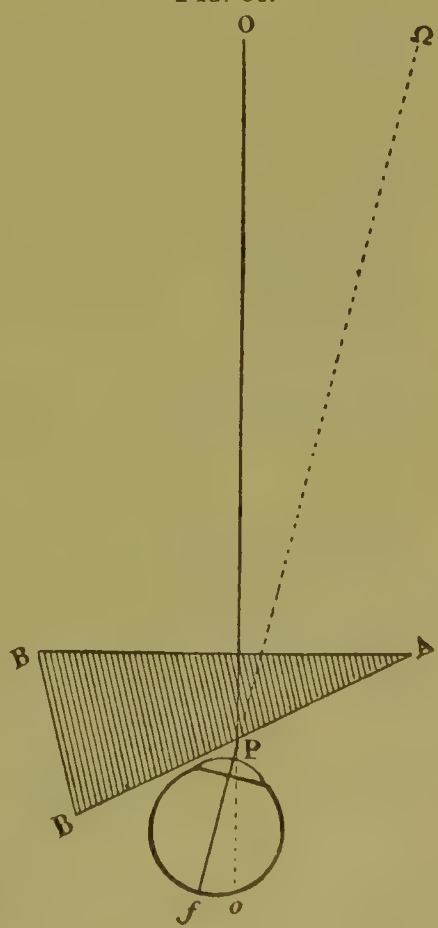
| | | |
|---|---|---|
| 30° = 18 cm. | 45° = 105 cm. | 55° = 141 cm. |
| 35° = 54 cm. | 50° = 124 cm. | 60° = 156 cm. |
| 40° = 82 cm. | | |

PRISMS.

The degree of the strabismus may be determined in still another way, —that is to say, by means of *prisms.*

A prismatic glass deflects towards its base the luminous rays which traverse it, in such a way that, to an eye looking through it, objects appear displaced towards the summit of the prism.

FIG. 30.




Thus, in Fig. 30, the ray *OP*, which comes from the luminous point *O*, instead of continuing its course in a straight line towards *o*, is, by the prism *BAB*, deflected towards *f*,—that is to say, towards the base *BB* of the prism,—as if it came from the point *Ω*.

Thus *oPf* is the *angle of deviation* of the prism. This angle is equal to the angle *OPΩ*, since *Po* is the prolongation of *OP*, as *PΩ* is that of *fP*.

This property of prisms may be utilized to direct upon the fovea centralis of a deviated eye the luminous rays which would not otherwise reach it.

Thus, if the eye in our figure be directed towards *Ω*, instead of being directed towards

*O*, the prism *BAB* will cause to fall on the fovea centralis *f* the image of *O*, which otherwise would be formed upon an excentric point (*o*) of the retina.

Now, if *O* is the object fixed by the sound eye, and $\Omega$ the point towards which the strabotic eye is directed, $OP\Omega$ is the *angle of strabismus*, and that angle is equal to the angle of deviation of the prism *BAB*.

This prism is called the *correcting prism;* for, by causing the image of the fixation-object to fall upon the fovea centralis of the affected eye, it brings about the fusion of the two images, it puts an end to the diplopia, and corrects, so to say, the strabismus.

Hence the correcting prism also gives the measure of the strabismus. That is one of the reasons why it seems logical to us to inscribe upon the prism the angle of the deviation which it produces. The diplopia corrected by prism No. 10 will indicate, then, a strabismus of 10°. And if we have ascertained, by some other method, the angle of a strabismus,—an angle of 8°, for instance,—we shall know that it is corrected by prism No. 8.[1]

It is true that, since the Washington Congress, two other propositions as to the method of numbering prisms have been made. The first is that of Dr. Dennett,[2] who would take as a limit of measurement of prismatic refraction the tenth of the *radian*, which he calls "centrad." The other proposition comes from Mr. Prentice.[3] This author would take as the measure of a prism the tangent of the deviation for a radius of one metre. His unit is a prism which at this distance produces an apparent displacement of one centimetre: he calls it the "prism dioptre."

This is not the place in which to enter into the discussion of these two methods, both of which are very ingenious. Their advantages are pointed out by their authors in the publications cited. In spite of that, and notwithstanding a most flattering letter which the American Ophthalmological Society has done me the honor to address to me, by its eminent Secretary, Dr. Samuel B. St. John,[4] in favor of Dennett's system (a letter to which I am happy to make here a respectful response), I still find it more practical and

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[1] Up to the present, prisms have been numbered according to their *angle of opening* (*BAB*, Fig. 30). This principle of the numbering of prisms is neither logical nor practical; for the angle of opening scarcely interests us, as it does not indicate the deflective action of the prism. This action, with which alone we are concerned, depends, indeed, not merely upon the angle of the prism, but also upon the material of which it is made. The angle of deviation of a prism is about one-half of its angle of opening, in the case of ordinary spectacle glass. It increases and decreases with the index of refraction of the glass.

It was in order to obviate this inconvenience, and to facilitate, for the oculist, the employment of prisms in the measurement as well as in the correction of the motor troubles of the eye, that the section in Ophthalmology of the International Congress of Medical Sciences at Washington (1887) charged Drs. Edward Jackson, Swan M. Burnett, and myself to propose to the next International Congress to number prismatic glasses according to the angle of the deviation which they produce. I acquitted myself of this mission at the International Ophthalmological Congress at Heidelberg in 1888, as well as at that of Medical Sciences at Berlin in 1890, and I have not ceased to do my best to further this useful reform. See also E. Maddox, Journal of Anat. and Physiol., xxi. p. 32.

[2] William S. Dennett, Transactions of the American Ophthalmological Society, 1890.

[3] Charles F. Prentice, The Metric System of Numbering and Measuring Prisms, Archives of Ophthalmology, iii., 1890.

[4] July, 1890.

simple to *number the prisms according to their angle of deviation*, just as in strabometry, in measuring the excursions of the eyes, in designating the angles Kappa, Alpha, Gamma, etc.

The centrad may have its advantages in physics, but physicists have not yet adopted it. How could we assume this reform, we who have only distant relations with the exact sciences? This reform, in order to be logical, would include, moreover, a considerable change in the graduation of all our apparatus and instruments—perimeter, ophthalmometer, etc., all divided in degrees.

Strabometry with the aid of prisms is, as we have pointed out,[1] subject to a slight error. The correcting prism may prove to be a shade weaker than the angle of strabismus. This fact is explicable in the following way. When a prism has brought the double images to within a short distance of each other, the tendency to binocular and single vision is powerfully awakened and provokes a most energetic contraction of the paretic muscle in order to realize the fusion of the two images. The instinctive feeling of the impossibility of attaining fusion prevents the patient from making that effort while the diplopia is still too great.

The same fact shows itself also in a very striking way in Fig. 7. The curve, which represents the diplopia of a person affected with paresis of the left external rectus, is regular up to the thirty-fifth degree at the right. There, it descends suddenly, to become fused with the horizontal line. This means that the separation between the double images, having diminished gradually during the gaze towards the healthy side, becomes suddenly *nil*, as soon as the images are so near to each other that a slight effort of the paretic muscle suffices to bring about their fusion.

In order to avoid this error, one need only choose, as correcting prism, that prism which causes and maintains, without effort, the fusion of the double images.

The three methods of strabometry—first, by means of the *corneal reflex;* second, by means of *tangents;* and, third, by means of *prisms*—are the most rational and most practical ones with which we are acquainted. Being each based upon a principle differing from that of the others, but all tending to the same end,—the determination of the *angle* of the strabismus,—they exercise an excellent control over each other. Hence we recommend them to all who wish to determine the degree of deviation of an eye with a precision sufficient and easy to obtain in practice.

These methods of strabometry are rational, because they measure the angle of the deviation of the eyes and express it in *degrees.*

It is incomprehensible that any one should ever have attempted to express a deviation, a rotation of the eye,—that is to say, an essentially angular value,—by linear measures. Nevertheless, we still hear of a strabismus of so many millimetres or lines, as if the eye, that has turned around its centre of rotation, had been displaced along a line. This is quite as absurd as if one should wish to express exophthalmia or any other displacement of an eye by means of angular degrees.

[1] Landolt, De la Strabométrie, Annales d'oculistique, 1875.

Indeed, logical in this lack of logic, the author of one of the principal works on this subject has substituted millimetres for the angle of strabismus and expresses the difference of the level of the eyes by degrees. Let us hope that the future generation will do justice to these aberrations which we have never ceased to combat.[1]

### DETERMINATION OF THE MONOCULAR FIELD OF FIXATION.

The degree of the deviation does not suffice to give a correct idea of the strabismus; we must further know the field of excursion, or *field of fixation*, of each of the eyes. This is true not only for paralyses of the ocular muscles, but still more for non-paralytic strabismus, with which we shall later become acquainted.

The field of fixation is most simply determined by aid of the perimeter, at the centre of which is placed the eye to be examined, the other being covered by a bandage. The eye is made to follow a fixation-object (letter, figure, printed word, or the like) which is moved, in all directions, along the arc of the perimeter. The degrees at which the object ceases to be seen distinctly, correspond to the limits of the excursions of the eye. These degrees may be inscribed on the same scheme as is used for the field of indirect vision. Their union gives the field of fixation. (See Figs. 12, 14, 19, 21, 22, 23, and 24.)

In order that this somewhat primitive experiment may be sufficiently exact for practice, it is necessary that the patient's head be immobile, otherwise its rotations will be added to those of the eye, and vitiate the result of our investigation. The head could not be more solidly or more simply fixed than by the teeth, which form part of the skeleton. For this purpose we use a horizontal strip of soft wood, screwed over the support of our perimeter. It is wrapped in a bit of white paper, and the patient is directed to seize solidly, between his molars, the right side or the left side of the mouth-piece, according as one wishes to examine the left or the right eye, which thus finds itself at the centre of the perimeter.

Instead of answering by words, which would cause a relaxation of the bit, the patient indicates by signs made with his hand, whether or not he sees the object distinctly.

Another precaution is to select fixation-objects of such a kind that the patient can distinguish them by direct vision only. If, for instance, he still read the word by the aid of a part of the retina distant from the fovea centralis, this angle of excentricity would unduly increase the field of fixation. However, this is less to be feared, as a matter of fact, than is the opposite. It is noticeable, indeed, that sight decreases in the extreme directions of the gaze; in other words, that the object of fixation appears indistinct even to a normal eye, when the latter approaches the limits of

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[1] Snellen und Landolt, Strabometrie, Graefe und Saemisch, Handbuch der gesammten Augenheilkunde, iii. S. 235, 1874.

its field of fixation. Hence it is desirable to select an object which is readily distinguishable, but which, notwithstanding, cannot be recognized by indirect vision.

When the vision of the eye under examination is very feeble, or the patient lacks intelligence, the field of excursion is to be determined *objectively* by the aid of the corneal reflex. A small lighted candle is placed at the zero-point of the arc, and the patient is made to fix it. By looking above or below the arc, as in strabometry (p. 44), one then places one's own eye so that the reflex of the flame appears just at the centre of the pupil of the examined eye. Then one moves the candle along the arc, having the patient's gaze follow it, and accompanies it with one's own eye until the reflex leaves the pupillary centre,—that is to say, until the examined eye can no longer follow the object. This point corresponds to the limit of the field of excursion.

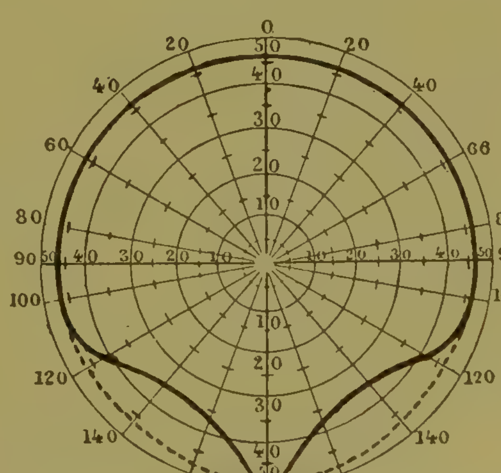
The field thus determined is almost always a little more extended than the subjective field of fixation. The explanation of this fact is to be found in the lessening of sight which we have just pointed out in the extreme deviation of the gaze.

Just as in the case of the angle of strabismus, it is best, in order to be exact, to take into account the angle Kappa in the objective mensuration of the field of fixation.

### DETERMINATION OF THE BINOCULAR FIELD OF FIXATION.

The most important thing to determine in every case of strabismus—indeed, in every case of a trouble in the motility of the eyes—is the *binocular field of fixation,* the extent of the space over which the lines of sight of the two eyes can meet in the same point of fixation. This experiment should be made at such a distance that the convergence may be disregarded. We use for this the mural division which we have described in the discussion of subjective strabometry. We have marked for this purpose upon the wall the tangents of the multiples of 5° in nine meridians, separated by 20° from each other, also the meridians inclined at 45°. (See Fig. 31.)

Fig. 31.




Normal binocular field of fixation.

The person to be examined is placed before this division in such a way that his eyes correspond to the centre of the imaginary hemisphere whose projection is inscribed on the wall and floor. The head is fixed by means of a dental strip supported by a solid pedestal. Then one moves, along the principal meridians of the chart, a lighted candle, which the patient follows with his eyes until he

commences to see it double. The point at which this diplopia appears constitutes the limit of the field of binocular fixation in each given direction. This is recorded on a scheme like that used for the record of the monocular field.

The perception of the diplopia is favored by a colored glass, which is best held by the patient himself before one of his eyes.

The full line in Fig. 31 corresponds to the normal field of *binocular fixation* of the author. The pointed curves at the left and right of the lower part of the figure are nothing else than the infero-external limits of the *monocular* fields of fixation. The nose prevents this space from being dominated simultaneously by both eyes.

## ETIOLOGY OF THE PARALYSES OF THE MOTOR APPARATUS OF THE EYES.

The affections which are capable of causing paralysis of the motor nerves of the eyes are numerous by reason of the long course of these nerves, their divers relations with adjacent organs, the relatively considerable extent of their central and peripheral expansions, and the multiplicity of the alterations of which these organs and regions are susceptible.

Following these nerves from their intra-hemispheric origin to their orbital termination, four principal groups may be distinguished in the paralyses of the motor nerves of the eye:

First, paralyses from lesions of the *intra-cerebral course of these nerves;* second, paralyses from lesions of their *protuberential nuclei;* third, paralyses from lesions of these nerves in their course after their apparent origin,—that is to say, *after their issue from the brain up to their entrance into the orbit;* fourth, paralyses from lesions of their *intra-orbital ramifications.*

The cerebral affections as well as the general maladies which may beget paralyses of the motor nerves of the eyes are treated of in special articles of this System. We refer our readers to these works of our eminent collaborators.[1]

Here we have to deal only with the third and fourth groups,—that is to say, with the lesions to which these nerves are exposed after having left the brain.

The changes which the motor oculi, the abducens, and the patheticus may undergo in their path at the base of the brain are rarely essential. It is more commonly external causes which influence them.

The principal one is *pressure* exerted upon their trunks. This may be due to *hemorrhages,* spontaneous or traumatic, which occur in the cranial cavity; to *exudations* accompanying *meningitis;* to *tumors* which originate within the *cerebral substance,* or in its *envelopes,* or in the *bony tissue* of the cranium. At times these nerves participate in an inflammatory process

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[1] See also Landolt and Eperon in De Wecker and Landolt, Traité complet d'ophthalmologie, iii. p. 840.

developed in their vicinity (*meningitis, progressive general paralysis*,[1] and *thrombosis of the cavernous sinuses*). Finally, they may be destroyed or compressed at some point of their course by traumatism (*fracture of the cranium*).[2]

Certain *tumors* of slow growth and of small volume may produce very localized paralyses of the motor nerves of the eyes. This is especially the case in *aneurisms* developed on the arteries which are in intimate relations with these nerves. Thus, Rauchfuss[3] has observed a paralysis of the left motor oculi produced by an *aneurism* of the left posterior cerebral artery, the size of a pea, which pressed upon this nerve.

Nieden has reported an observation of an isolated paralysis of the right patheticus due to the pressure exerted upon the nerve by the pineal gland, which had undergone *cystic degeneration*.

*Syphilomata* may produce the same result.

Tumors of the cerebellar fossa, those of the pons Varolii, those of the petrous bone, and those which have their origin in the walls of the cavernous sinus, are those which most expose the motor nerves of the eye to pressure. The last two classes generally involve also paralysis of the other bulbar nerves.

The motor nerves of the eyes are so liable to be damaged by *traumatisms* of the cranium, that paralysis of these nerves is sometimes the only symptom present when the injury has not been sufficiently severe to cause grave changes in the intra-cranial mass. Thus, *fractures of the base* are frequently accompanied by paralytic strabismus, especially those which concern the petrous bone. M. Panas has called attention to the fact that in such a case the paralysis of the external motor is more frequent than that of the third and fourth pairs. This is due, according to that author, to the peculiar relations of these different nerves with that region of the cranium.[4]

In contradistinction to paralysis of the common motor oculi of bulbar origin, that which is produced by pressure or rupture of its trunk at the base of the brain is, if not always absolute, at least frequently total,—that is to say, it extends to all the muscles innervated by the affected nerve.

Our fourth group comprises *paralyses due to lesions of the terminal orbital branches of the motor oculi nerves.*

At their entrance into the orbit, through the sphenoidal fissure, the nerves which supply the muscles of the eye are still very much exposed

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[1] Magnan, Note sur la sclérose des nerfs optiques et des nerfs moteurs de l'œil dans la paralysie générale (Gazette Médicale de Paris, No. 44, 1877).

[2] For the different symptoms which may accompany the basal paralyses of the motor nerves of the eyes, and for the peculiar value of these paralyses as concerns the localization of a cerebral lesion, see Nothnagel, Topische Diagnostik der Gehirnkrankh., 1879.

[3] Rauchfuss, Zur Casuistik der Hirnembolie, Petersb. Med. Wochenschr., No. 7, 1878.

[4] Panas, De la paralysie du nerf moteur oculaire externe consécutive aux traumatismes du crâne, Archives d'Ophtalmologie, i. p. 3, 1880–81; and Genouville, Fracture de la base du crâne avec paralysie du nerf moteur oculaire externe, Archives d'Ophtalmologie, February, 1883.

to the injurious influences which may result from lesions of the skeleton (*periostitis*, *gumma*, *fractures*).

Even in the interior of this cavity, they appear to be subject to inflammation of their neurilemma in a spontaneous way (perhaps a part of the motor paralyses of the eye *a frigore* is due to an inflammation of the muscular tissue itself), oftenest under the influence of cold (*rheumatismal paralyses*), or consecutively to *inflammation of the cellular tissue of the orbit.*

Thus, Landesberg[1] has observed a paralysis of almost all the muscles of both eyes, without any cerebral symptom, in consequence of exposure to cold. The cure was perfect at the end of a short time.

Von Hippel[2] has seen a complete paralysis of the muscles of the eyes with symptoms of inflammation and œdema of the orbital tissue, which he attributes to a periostitis of the sphenoidal fissure.

Perhaps it is to such changes that the paralysis sometimes observed in cases of *dental caries* (Baumeister has related a case of this sort) or of acute *auricular rheumatism* must be attributed.

We have observed, with our clinical assistant, Dr. Gygax, a very strange case of *paresis of the inferior rectus*, caused by an *inflammation of the mucous membrane of the antrum of Highmore.* There was, at the same time, a certain degree of protrusion of the globe; but the proof that it was not merely a question of displacement of the globe by pressure was to be found in the very distinct symptoms of paresis of the inferior rectus. Thus, there was strabismus sursum vergens of 10°, with slight divergence of the eye and corresponding vertical diplopia, increasing when the patient looked downward and to the affected side, diminishing when he looked in the opposite directions.

It is evident that a *tumor* at all voluminous, developing itself in the narrow space which the orbital terminations of the motor nerves of the eye occupy, has for its effect their compression or annihilation. But in such a case the lesions of the nerves are hidden in those of the muscles which they innervate.

Paralysis of *orbital* origin may be partial and localized, but it is oftenest general. It is usually accompanied by periorbital pains. Movements of the globe, and pressure from before backward on the closed eye, are equally painful when there exists an inflammation of the cellular tissue of the orbit. The latter is generally accompanied by chemosis, and there is a certain degree of exophthalmos, still more accentuated when caused by a neoplasm developed in this tissue.—Finally, the internal and external frontal nerves are often affected at the same time. Hence there will be found, in many cases of this kind, a certain degree of anæsthesia of the skin of the forehead.

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[1] Landesberg, Ueber doppelseitige Augenmuskellaehmungen, Berliner klinische Wochenschrift, SS. 645–648, 1874.

[2] Von Hippel, Laehmung aller Muskeln eines Auges, Bericht Ophthalmologische Klinik, Giessen, 1879–1881, S. 22.

Fraenkel[1] describes a sort of *serous degeneration of the ocular muscles* in progressive anæmia. This lesion, moreover, does not appear to have caused strabismus.

Mention should also be made of the *congenital paralyses* of the ocular muscles. They are not so rare as seems to be generally supposed. Scharpinger[2] relates a case of congenital paralysis of the sixth and seventh pairs (abducens and facial).

We have ourselves observed several cases of *congenital ptosis.*

It is possible that many cases of strabismus in young children, which are usually considered as non-paralytic, because the symptoms which characterize paralysis of the ocular muscles are not very pronounced, are properly referable to congenital paresis. If diplopia is lacking, it is simply because binocular vision did not exist at the time of the production of the paresis. The association of the movements of the two eyes likewise undergoes, in such a case, changes capable of interfering with the classic aggregation of the symptoms of muscular paralyses.

## ETIOLOGICAL DIAGNOSIS OF THE OCULAR PARALYSES.

All the etiological circumstances which we have just enumerated generally present themselves at once to the mind of the physician, in the presence of a muscular paralysis of the eye. A careful anamnesis and examination will enable us to eliminate the majority of them, by directing our attention more especially to certain ones among them. Generally, affections of this kind, when they arise in young and apparently healthy individuals, when not due to traumatism, are of rheumatic or syphilitic origin. Hence one ought to suspect the presence of *syphilis* in such a case, and inquire concerning the patient's morbid antecedents.—If the affection began subsequent to exposure to cold, and is accompanied by frontal and periorbital pain with painful sensations when the eyes are moved, it is likely to be of *rheumatismal* origin.—If neither of these two causes appears to be in action, it is best to seek for symptoms of *tabes dorsalis,* such as cubital anæsthesia, cardialgia, the decrease of sensibility, shooting pains, difficulty in micturition and defecation, etc., the abolition of the patellar reflexes, and—a symptom much more characteristic—the immobility of the pupil under the influence of light, its contraction accompanying convergence. (Argyll-Robertson.)

The urine ought always to be examined, in order to learn whether or not it contains sugar.

If all these researches remain without result, we may investigate the possibility of the paralysis being due to *poisoning by lead, diseased meat, tobacco, alcohol,* or by the other injurious substances which we have mentioned above; finally, *hysteria* has to be taken into account.

The nature of the paralyses which follow *diphtheria* will soon be recog-

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[1] Fraenkel, Deutsches Archiv für klin. Med., v. 20, H. 5, 1877.

[2] Scharpinger, New Yorker medicinische Monatsschrift, Ueber angeborene beiderseitige Pleuroplegie, December, 1889.

nized, thanks to the anamnesis. The latter, however, ought to be peculiarly exact, considering all the details. It may happen that paralyses are produced in consequence of an angina which is without any apparent gravity and which may have escaped the patient's notice.

Those which may be dependent on a *circulatory disturbance of the nervous centres* are generally accompanied by the cerebral symptoms customary to such lesions,—cephalalgia, a feeling of heaviness of the head, buzzing in the ears, indisposition for work, somnolence, etc. One is to seek carefully the causes which may bring about these troubles (alterations of the digestive functions, constipation, overwork, over-indulgence at table or in alcoholic beverages, intestinal parasites, and febrile processes).

If the paralysis be due to an *anatomical alteration of the intra-cranial tissue*, it cannot fail to be accompanied by other symptoms, such as hemiplegia or hemiparesis, monoplegia, facial paralysis or paralysis of the other bulbar nerves, more or less complete abolition of the cerebral functions, vomiting or epileptiform seizures.

Finally, *hysteria* will betray itself by its well-known signs,—plaques of anæsthesia or complete hemianæsthesia, ovarian pain, the bulbus hystericus, determining pressure-points, convulsive seizures, more or less marked unilateral amblyopia with diminution of the retinal functions, and narrowing of the visual field for white and for colors, as we pointed out in 1875.[1]

We found and demonstrated to our students, several years ago, a characteristic symptom by means of which to distinguish paralytic ptosis from an occlusion of the eye in consequence of a spasm of the orbicularis. In the case of paralysis, the patient seeks, as is well known, to substitute a contraction of the frontal muscle for the insufficient action of the levator palpebræ superioris. The skin of the forehead is therefore folded, and the eyebrow of the affected side is higher than that of the healthy side. On the contrary, when there is spasm of the orbicularis, the eyebrow of the affected side is lower than that of the healthy side.

At another time, in connection with a double ptosis of hysterical nature, I noticed the following fact. I told the patient to look at my eyes. She leaned her head backward in order to be able to see under her lowered lids. I then gently inclined her head forward, at the same time enjoining her to continue to fix my eyes. In proportion as her head was lowered, her eyes and eyelids were raised. Finally, when her chin almost touched her chest, it was found that her upper lids were so much raised that they were almost hidden under the eyebrows. This was an irrefutable proof that it could not be a question of paralysis of the levators; otherwise, at the slightest attempt to lower the head the pupils would have been hidden under the inert lids.

As to the diagnosis of the *seat of the lesion*, it can be made accurately

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[1] Landolt, De l'amblyopie hystérique, Archives de physiologie normale et pathologique, 1875, p. 624.

only if all the peculiarities of ocular paralysis are carefully taken into consideration along with the other concomitant symptoms. We have already seen that the partial paralyses of the common motor oculi, especially those wherein the pupil and the accommodation perform their functions normally, very probably have a nuclear origin; while changes in the nervous cords at the base of the brain and in the orbit almost necessarily entail a total paralysis of this nerve.

## PROGNOSIS, DURATION, COURSE, AND TERMINATION OF PARALYSES OF THE OCULAR MUSCLES.

The duration, evolution, mode of termination,—in short, the prognosis,—of a motor paralysis of the eye naturally all vary according to the cause which has provoked it. If it be due to a curable affection, the nerve will be able, little by little, to resume its functions if, at the same time, the interruption has not been too prolonged, and if its fibres or the ganglion-cells of its nuclear centre have not undergone too profound alterations.

Thus it will be understood that paralysis due to a simple *circulatory disturbance* of the encephalon or of the isthmus of the encephalon may be easily curable, as soon as one shall succeed in suppressing the morbid influences which are in action. The same is true of *hysteria*, in which the nervous paralyses depend on functional lesions.

Paralysis consequent upon *hemorrhage in the cerebral substance* is likewise susceptible of cure when the effusion has only pressed upon the nerve-cord. It is equally so in cases where a small hemorrhage (the rupture of an aneurism) has not produced a too extended destruction of the ganglion-cells of the nuclei or when it has acted simply by reflux and compression on its vicinity. In hemorrhage into the hemispheric substance, at the time when the secondary contractions of the limbs take place,—contractions which announce the irreparable destruction of the internal capsule,—the other phenomena of compression ameliorate, the unilateral or ocular paralysis entirely disappears.

This is not the case, of course, when an abundant and sudden hemorrhage has reduced to a pulp the nervous substance of the protuberantial nuclei, when they have been involved in a focus of softening of the brain, of acute or chronic (sclerosis) encephalitis, or when any other tumor than a syphiloma compresses or abolishes them.

It is, as a rule, wrong to make a favorable prognosis concerning the tabetic paralyses of the motor nerves of the eyes; although, as we have said, these paralyses readily disappear of themselves or under the influence of treatment, they may return and become permanent. It ought not to be forgotten, moreover, that they may persist from the outset in many cas s, and that they have a pronounced tendency to invade all the muscles of the eyes, like those that accompany progressive bulbar paralysis. However, Benedikt tells of having seen such paralyses cured by very prolonged

electrization. The same remarks are applicable to paralyses depending upon sclerosis plaques.

Pressure upon the nervous trunks at the base of the cranium or in the orbit by meningitic exudations, by thrombosis of the cavernous sinus, by a tumor, or by a bony particle, necessarily entails an unfavorable prognosis. The prognosis is doubtful when the nerve lesion is of traumatic origin. It may happen in such a case that it is a question simply of compression of the nerve by a hemorrhagic effusion, whose resorption brings about the restoration of the conductibility; but too often the nervous trunk is partially or wholly destroyed by the injurious cause, or is included by a fracture callus, and irremediably lost.

The prognosis of paralysis due to *syphilis* is not so serious, because of the amenability of these lesions to treatment. Although disturbances of motility of specific origin usually improve under proper treatment, yet it is well not to abandon one's self to too great delusions as concerns the result of such treatment. External ophthalmoplegia, among others, or bulbar paralysis of syphilitic nature does not always improve under the influence of such medication.

The same remarks are applicable in the case of *lead poisoning*. *Diabetic* paralyses and those of *diphtheritic* origin are generally benign, and get well with the lapse of time, inasmuch as the ocular disturbances are not complicated by lesions of other nerves essential to life, such as those of respiration and circulation.

So-called *rheumatic* paralyses may get well entirely and rapidly. They persist, on the contrary, with great obstinacy, or become incurable, when they are long neglected, or when the inflammatory lesions of the nerves have been peculiarly profound.

The same is true of the paralyses which accompany *migraine*. Paralyses associated with migraine—that is to say, those which manifest themselves in persons subject to this malady—generally disappear promptly at the end of a few weeks. But they almost always return, sometimes affecting another muscle, sometimes even the other eye, or attacking both eyes simultaneously. Hence if the immediate prognosis is not unfavorable, the ultimate prognosis is far from being altogether good, and so much the more since migraine may be the prodrome of a serious cerebral affection.

The *hysterical* paralyses (and contractions) of the ocular muscles comport themselves like the other morbid manifestations to which hysteria gives rise. They may persist during entire years with an obstinacy defying all imaginable therapeutic efforts; at other times they disappear as if by magic. But, so far as our experience goes, they are rather resistant to, than susceptible of, improvement.

The ocular muscles being in great part inaccessible to the direct application of electricity, we are deprived of this precious means of diagnosis and of the information which it might furnish us with reference to the prognosis. However, a careful consideration of all the facts which we

have discussed—the anamnesis of the patient, the examination of the functions of his cerebro-spinal system, and the general phenomena which may be presented—permits of our detecting, with relative certainty, the seat of the paralysis, its nature, and the consequent prognosis.

When a muscular paralysis of the eye is curable, it is seen to amend more or less rapidly, whether in a spontaneous way or as the effect of appropriate treatment. The distance between the double images diminishes by degrees, the domain of single vision broadens at the expense of that of the diplopia, and the deviation of the eye becomes less striking. The head assumes a more natural position, the excursions to the side of the paralyzed muscle gain in extent and ease, and the field of fixation increases in this direction. It may thus happen that at the end of a few weeks the cure becomes complete. There persists, at most, a slight diplopia in the extreme direction of the eyes to the side of the paralyzed muscle, the last vestige of which is at length effaced.

But it may also happen that, after having made a certain amount of progress, the amelioration remains stationary, and perfect recovery is obtained only by an extremely long and persevering treatment.

Finally, there are many cases in which the paralytic deviation becomes permanent. The visual troubles which accompany it amend, however, little by little. The diplopia, with the vertigo that it produces, becomes less troublesome; the competition between the two visual fields is overcome by virtue of the exclusion of one of them. The fixing eye (sometimes the one which is paralyzed) remains the sole master of vision. As to the other, it becomes accustomed to its new position of equilibrium, and succeeds finally in accurately projecting its retinal images. The muscle or muscles which keep the eye in its vicious position, being permanently shortened, undergo nutritive modifications tending to diminish their real length in the absence of normal contraction. They become the seat of what is called *secondary contracture.* (Von Graefe.)

Secondary contracture develops rather rapidly, and it is necessary to combat it, even in cases which are certain to recover. According to what we have just said, it is developed as well in the sound eye, when the affected eye is used for fixation, as in the affected eye, when this deviates, which is usually the case. It may persist after a complete return of the conductibility of the nerve, and give rise then to a sort of concomitant strabismus, accompanied by symptoms similar to those of the paralytic strabismus which gave rise to it. Their distinction sometimes presents difficulties, which the examination of the fields of fixation of the secondary deviation and of the projection may not always overcome.

The secondary changes in the muscles, following paralysis of the nerves which supply them, are as yet very little known. It seems, according to Von Graefe,[1] that the paralysis of an ocular muscle does not certainly entail

[1] Mauthner, Nuclearslaehmung, S. 808.

its atrophy. On the other hand, Westphal has observed fatty degeneration of the muscles in consequence of nuclear paralyses.[1]

## THE TREATMENT OF PARALYTIC STRABISMUS.

The treatment of recent paralytic strabismus has two principal indications to fulfil: first, to dissipate the visual disturbance which troubles the patient, until he is completely cured; second, to promote this cure, at the same time preventing the formation of secondary contractures of the antagonists of the paralyzed muscles.

In certain cases a surgical operation may be required to restore single binocular vision, or at least to remedy the disfigurement of the strabismus, in the principal direction of the gaze.

The first object may be achieved in different ways. The simplest means, when the deviation is considerable and the diplopia very marked, consists in *covering one of the eyes with an opaque screen* or with a ground glass. It will naturally be the affected eye which is subjected to this occlusion, whenever choice is possible. The secondary deviation being, indeed, more considerable than the primary, it would be very inconvenient to abandon the sound eye to this deviation, which might occasion a secondary contracture of the muscle or of the group of muscles which produces it. However, we may be forced to do this, when the visual acuity of the sound eye is much inferior to that of the diseased one. The choice of the fixing eye is almost always instinctively made by the patient, especially when both eyes are the seat of a motor paralysis.

When the deviation is slight, and the double images are consequently very near each other, the paresis may be remedied with the aid of *prismatic glasses.*

We have, in the section on strabometry, become acquainted with the action of these diopters.[2]

By changing the direction of luminous rays, they may cause the image of the object fixed by the healthy eye to fall upon the fovea centralis of the deviating eye, and thus bring about fusion of the two images,—that is, binocular vision.

Now, the prismatic deviation taking place towards the base of the prism, the apex of the prism has to point to the side towards which the eye deviates. In convergent strabismus, the apex of the prism is towards the nose, in strabismus sursum vergens, the apex is upward, etc.

It is necessary, moreover, that the optic effect of the prism be equal to the deviation of the eye. A strabismus of 10° requires a prism of 10° of deviation; a strabismus of 20°, a prism of 20°, etc. (See page 49.)[3]

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[1] Westphal, Jahresbericht ueber die Leistungen und Fortschritte im Gebiete der Ophthalmologie, Tübingen, 1884, SS. 343, 605

[2] According to Monoyer, transparent substances cut so as to produce a dioptric effect are called "diopters."

[3] We call a prism which produces a deviation of 20° a prism of 20°, etc.

Here we notice at once one of the weak points in the use of prisms in paralytic strabismus. We know, indeed, that the deviation increases to the side of the affected muscle, while it diminishes in the opposite direction. Hence the same prism can neutralize the diplopia produced by the strabismus in but one direction. This is the case, indeed, in complete paralysis. But in this case prisms are not of much use, for a cogent reason, with which we shall presently become acquainted.

In muscular paresis of slight degree, on the contrary, prisms render great service. When the deviation is a horizontal one, the patient readily remedies the insufficiency, or the excess of action, of a prism, by an increase or a diminution of his convergence, or by a slight rotation of his head.

This rotation will be specially useful to him in the case of vertical deviations. Let us suppose, for instance, an eye directed upward by three degrees, in consequence of the paresis of a depressor. A prism of 3°, with apex upward, will bring the images to the same level when the gaze is straight ahead. If the patient lowers his eyes to look downward, the diplopia will reappear, because the affected eye cannot follow the healthy one, on account of the insufficient action of the paretic muscle. But the patient will soon learn to lower the head in order to escape this embarrassment. He will raise it, on the contrary, if, elevating his gaze, the prism should prove to be too strong, because the strabismus diminishes in this direction.

We have, moreover, pointed out, when discussing strabometry, the fact that when two images are near enough to each other—in other words, when the retinal image of the deviated eye is formed near enough to the fovea centralis—fusion is often spontaneously accomplished, so that a prism whose action is somewhat less than the degree of the strabismus may, notwithstanding, suffice to correct it.

Of course, a prism could not remedy the inclination of the image belonging to an eye which has undergone a pathological rotation around its antero-posterior axis. But here again observation proves that, when the images are formed in each eye on the fovea centralis, or very near it, the obliquity of one of them is easily neutralized, whether by an inclination of the head or by the combined action of the muscles of both eyes.

But the use of prisms, so valuable for the correction of strabismus, is, unfortunately, limited by certain other optical effects which are peculiar to them (coloration of outlines, changes in the form and relief of objects), and also because of the weight of such glasses, as soon as they exceed a few degrees. Thus it is that a prism producing more than three degrees of deviation is worn with difficulty.

It is true that both eyes may be furnished with prisms, so that, instead of placing a prism of 6°, with its apex nasal, before the left eye, this eye being affected with convergent strabismus, a prism of 3° may be placed

before each eye. Each of the prisms ought, necessarily, to have its apex directed towards the nose.

The same is true in the case of divergent strabismus. Two prisms whose apices are directed towards the temples, when placed before the two eyes, have the same effect as a single prism whose strength is equal to the sum of that of the other two, this one being placed before one eye.—The same thing occurs again for vertical deviations. Instead of placing the prism of 7°, with its apex upward, before a left eye which deviates upward, we may give three degrees to this eye and four degrees to the other; only, for this latter, the apex of the prism will be directed downward.

This distribution of the correction of the two eyes is particularly indicated in intermediate deviations. In such a case, the prism which corrects the horizontal diplopia may be placed in front of one eye, and that which corrects the difference in height, before the other.

It is self-evident that for persons who usually wear spheric or cylindric or combined lenses, we should utilize the two surfaces of the prism to correct the optical defect; in other words, the prism will be combined with the correcting glass of the ametropia.

The *symptomatic* treatment having been instituted, we must direct our attention to the restoration of the conductibility of the nerves, and of the contractility of the muscle. The means we employ must naturally vary with the *cause* of the paralysis. Rest, derivative medication, a regimen appropriate to the individual sanitary conditions, will happily modify those *cerebral changes* dependent upon anæmia, hyperæmia, plethora, etc.

In *syphilis*, specific remedies should be employed,—*e.g.*, a course of mercurial inunction or injection and iodide of potassium.

A suitable diet, along with the administration of Carlsbad or Vichy waters and of bromide of potassium, forms the proper treatment of *diabetes*.

*Lead poisoning* requires sulphur baths and iodide of potassium internally; *hysteria*, hydrotherapy, electricity, antispasmodics, and moral suasion.

If the paralysis be due to a *peripheral affection* of the motor nerves caused by cold, it may be well to oppose it with the customary antiphlogistics: local bleeding, diaphoresis by means of warm beverages or pilocarpine, moist packing, laxatives, and, if rheumatism assume an acute and general form, salicylate of sodium. Later, the application of a vesicatory has been recommended as favoring the complete resolution of the inflammation of the oculo-motor nerves.

Whatever may be the causal indication to be fulfilled, electricity is considered an important adjuvant. Stimulation of the nerve is ascribed to it, the stimulation of its nutrition and of its special function, the excitation of the contractility of the muscle, which prevents degeneration of its tissue.

The constant current and faradization are both employed under such circumstances. The mode of application varies much according to different

authors. Some advise acting directly on the great sympathetic by placing the negative pole on the superior cervical ganglion, the positive on some point of the orbital border close to the paretic muscle.[1] Others recommend the electrization of the affected nerve itself; they apply the cathode to the back of the neck, the anode around the orbit. The current ought generally to be weak (from four to six elements) and of a duration of from two to five minutes; sittings may be repeated on alternate days.

If it be desirable to use the induced current, it may be applied directly to the muscle. For this purpose, the electrode, in the form of a little flattened knob, isolated on one side, is introduced under the lids as far back as possible, and its free surface applied on the diseased muscle. Cocaine anæsthesia will facilitate this little manœuvre.

At regular intervals, the effect of the treatment is to be ascertained by the aid of careful strabometry. If our efforts are crowned with success, there will come a time when the deviation can be corrected by a prism weak enough to be permanently worn. This will hasten the cure by stimulating binocular fusion.

J. Michel proposed, in 1877, a treatment of muscular paralysis which may be called *mechanical*. This method consists in seizing the conjunctiva over the insertion of the paralyzed muscle by means of fixation forceps, and turning the globe forcibly as far as possible in the direction of the action of the muscle, to and fro, for a few minutes. Charles Stedman Bull[2] treated in this way, prior to 1887, twenty-one cases, of which eight were cured, six were improved, and seven remained stationary. Patients submit unwillingly to this method of treatment, which is undoubtedly painful.

We have already twice mentioned the tendency that most patients manifest to fuse the double images as soon as these are brought near enough together. This is the reason why the degree of strabismus determined by the correcting prism is generally less than that which results from the diplopia. The treatment of strabismus which may be called *orthoptic* is founded upon this observation. By some means, whether with the help of a prism or simply by means of rotation of the head, the two images are brought so near together that they may be fused. Then an attempt is made to maintain the fusion under less favorable circumstances. This is done either by diminishing the strength of the prism[3] or by turning the patient's head slowly in the direction opposite to the strabismus, while he still tries to maintain the fusion. In this way, the contraction of the paretic muscle is stimulated. It is true that in complete paralysis this device is unlikely to be successful.

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[1] See, with reference to this, Benedikt, Erb, Neumann (in Landolt, art. Strabisme, Dictionnaire encyclopédique des sciences médicales, p. 723, 1883).

[2] Transactions of the American Ophthalmological Society, 1887, p. 459.

[3] The double prism is of great use in such a case, because the diminution is made gradually and without the patient's cognizance.

One may also, with a view to varying the exercises, use for the same purpose our *stereoscope.* (Fig. 41.)

When this stereoscope is used, the eyes are adapted, by means of proper glasses (six or seven dioptrics for emmetropic eyes) to the images at the bottom of the instrument, and to the two images is given a separation in accordance with the divergence, convergence, or the difference in height of the lines of sight in the position of rest. For the usual length of the base line (sixty-four millimetres) this distance ought to exceed this value by about ten millimetres for seven degrees of divergence, and to be less by an equal amount for a like degree of convergence. Thus, if we have to do with a convergent paralytic strabismus of fourteen degrees, we should give to the two images a separation of about forty-four millimetres. If we are concerned with a divergent strabismus of ten degrees, the stereoscopic images are to be separated by about seventy-eight millimetres, etc. Under such circumstances, the stereoscopic fusion of the two images will be obtained. This result having once been achieved, we seek to get gradually the normal separation—that is to say, parallelism—of the lines of sight. One may even attempt to surpass this end, when one has succeeded in bringing about parallelism of the divergent lines of sight, and exercise them little by little in fusion in the convergent position.

### SURGICAL TREATMENT OF PARALYTIC STRABISMUS.

When the paralysis of an ocular muscle is not susceptible of medical cure, one is justified in having recourse to surgical means for remedying the annoyance which it causes the patient.

Personally, we have operated for the correction of paralytic strabismus oftener than is generally done. We have attacked cases which many would have considered as *noli me tangere* for surgery, and we have never found cause to regret our hardihood in this respect. It is true that our method of operating differs somewhat, as will be seen, from that generally in use.

Not being able to restore the contractility of a paralyzed muscle, operators have sought to weaken to an equal degree the associated muscle of the healthy eye, for instance, the internal rectus of the right eye in a case of paralysis of the external rectus of the left eye.

Some have even gone so far as to recommend the tenotomy of all the muscles acting in the horizontal meridian on the healthy as well as on the diseased eye, the purpose of this general tenotomy being to remedy the paresis of a single one of the muscles in question.

Thus, in a case of paresis of the left external rectus, the right internal rectus would be tenotomized; and, under the pretext of "equalizing the accommodation" in the two eyes, one would add to this tenotomy that of the left internal rectus, as if the accommodation of an eye depended on the strength of its internal rectus muscle.[1] Finally the externus of the healthy

[1] See A. Graefe, in Graefe und Saemisch, Handbuch der gesammten Augenheilkunde, ix. S. 82.

eye would be tenotomized, in order to restore the muscular equilibrium of this eye!

In my opinion, we ought to proceed quite otherwise. Given the inertia of the paretic muscle and, consequently, the difficulty of obtaining a field of binocular fixation of considerable extent, it is necessary, at least, that the field of fusion which we create shall be in the most useful direction for the individual,—that is to say, in the *median plane* for the muscles that act horizontally, in a plane *slightly lowered* for those that act vertically. This is true not only when binocular vision exists, but also when the operation is done solely with the cosmetic purpose of remedying the disfigurement caused by the deviation of the eye.

Moreover, I always abstain from weakening one of the muscles of the healthy eye. It is precisely in those cases for which the tenotomy on the sound eye has been most recommended that I practise this least: in cases of simple paresis.

There is always a chance of recovery from a paresis. On the contrary, the defect in motility resulting from the setting back of a muscle, far from recovering, is always on the increase. One thus incurs the risk of creating for the healthy eye a strabismus worse than that of the paretic eye. For this reason, we prefer to treat paralytic strabismus (as well as the non-paralytic strabismus) by muscular advancement rather than by tenotomy.

Let us take an extreme case, *a total paralysis of the external rectus of the left eye.* The patient sees single only in the extreme right part of his field of fixation. Diplopia, as well as the disfigurement which results from his convergent strabismus, annoys him; he wishes to be relieved of it.

In this case, I should perform an energetic advancement with resection of the external rectus on the diseased eye and a simple advancement of the same muscle on the other eye. If necessary, the tenotomy of the internal rectus of the diseased eye could be added.

Binocular vision and, with all the more reason, the desired cosmetic effect are thus obtained, not only for the "primary position," but also for raising and lowering the gaze.

It is evident that, as soon as the attention of the patient is directed to an object situated at the left, the convergent strabismus and the homonymous diplopia will reappear, because of the inertia of the paralyzed muscle. On the right side, on the contrary, there will be crossed diplopia, because the left eye only imperfectly follows the movement of its fellow in this direction. It lags behind because of the operative weakening of its internal rectus. There is divergent strabismus to the right, convergent to the left.

This is inevitable in a case of total paralysis, but certainly less annoying than the condition produced by tenotomy of the internal rectus of the healthy eye. He who watches carefully the effect of his operations knows very well that, in order that a tenotomy may counterbalance a muscular paralysis, an extreme setting back is required, such a setting back as is

obtained only by means of sutures applied over the antagonist,—the thread-operation, as von Graefe called it. Thus the eye is drawn out of its muscular funnel, and its mobility greatly damaged. This is a method which we absolutely condemn, for reasons to be given in the next section.[1]

Moreover, the patient soon learns to remedy, by the rotation of his head, the insufficiency of the movements of his left eye.

As to convergence, it certainly will be diminished by the tenotomy of the left internal rectus, but not nearly so much as by the thread-operations on the other eye. When the paralysis is not complete, no tenotomy is necessary to restore binocular vision. We may refer, for instance, to the case of a carpenter who could not do his work on account of a *paresis* of the *rectus externus* of his left eye. The paresis, indeed, had caused a *convergent strabismus* with homonymous diplopia of 12°. We performed *advancement of the external rectus of each eye*, *without tenotomy* of the antagonist, but resecting several millimetres of the paretic muscle. The result was perfect fusion. The patient could even overcome an abducting prism of one metre-angle ($\gamma = -1$); and as for the convergence, it exceeded the normal, in spite of the advancement of the abductors.

In another case in which the paresis of the external rectus had brought on a strabismus of only 5° 30′, advancement of this muscle alone succeeded in establishing absolutely harmonious binocular movements. Convergence, divergence, and even lateral excursion became perfectly normal.[2]

Our operative method is, *mutatis mutandis*, the same for *paralysis of the internal rectus*. Only, divergent strabismus always shows itself more resistant to treatment than convergent strabismus. Even the strongest advancement of the internal recti on both eyes suffices only to restore the parallelism of the eyes, but does not give the patient a converging power sufficient for his usual work. We may then venture to tenotomize the external rectus of the diseased eye. But this has always to be done with great precaution, otherwise one runs the risk of changing the divergent strabismus into a convergent one, or even of creating homonymous diplopia for distance without doing away with the crossed diplopia for near at hand work.

When the paralysis of an abductor or adductor muscle is incomplete, when there exists only *paresis*, with diplopia of but a few degrees, the surgeon's task is a much lighter and much more thankful one.

In such a case, it often suffices, as we have just shown, to give to the enfeebled muscle, by its advancement, a more favorable insertion in order to restore binocular vision over a very large, if not to the normal, extent.

The antagonist of the paretic muscle ought never to be tenotomized

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[1] See, also, Landolt, *compte rendu* of his clinique, 1878, Rapport sur le Strabisme, Seventh International Congress of Ophthalmology, Heidelberg, 1888; Archives d'ophthalmologie, 1880, i. p. 594; International Medical Congress, Washington, 1887.

[2] Landolt, Traitement chirurgique du Strabisme, Archives d'ophthalmologie, 1896, p. 420.

solely, as has been advised.[1] If the insufficiency of the tenotomized muscle equal that of an even moderately paretic muscle, it becomes a considerable source of annoyance, even in the lateral excursion, but especially in convergence, when the setting back has been performed on an internal rectus.

The tenotomy ought only to aid the advancement of the paretic muscle, and ought always to be executed very moderately, for *advancement increases the excursions of the eye, whereas tenotomy reduces them.*

Let us now consider the operative treatment of paralysis of the muscles which act vertically,—the *depressors* and *levators* of the eyeball.

It might be supposed that our task would present in this case insuperable difficulties. This would be true if we had to remedy all defects of direction and rotation to which paralysis of one of these muscles gives rise. Indeed, the obliques, as well as the superior and inferior recti, turn the eye, as it were, around three axes,—transverse, vertical, and antero-posterior. Now, even combined operations could not make good the troubles produced in these three directions. But, fortunately, experience has proved that the problem is not so complicated, and that we have in nature a powerful and benevolent aid, ready to fill up the gaps left by our always imperfect art, provided these lacunæ be not too great.

Let us again take a few examples and apply to them our principle of *correcting strabismus for the most useful direction of vision.*

Suppose a *paralysis of the superior oblique of the left eye.* There is a strabismus sursum vergens, slightly convergent, and, moreover, temporal rotation of the eye around the antero-posterior axis.

It has been advised[2] that this state of things be remedied by setting back the inferior rectus of the healthy eye, with a view to creating in this eye the same strabismus as exists in the affected eye. The right eye would thus be directed upward, to the right, and inclined towards the left, like its paralyzed neighbor. (See page 25, Paralysis of the Inferior Rectus.)

If by this means the retinal images of both eyes be made parallel and brought to the same level, it is done at the expense of the lowering of the gaze. Now, the lowering is precisely the "most useful direction of the gaze," that for which, following our rule, the strabismus ought to be corrected. Von Graefe's method adds to pathological elevation of the affected eye the operative elevation of the healthy eye, making the lowering of this latter eye as difficult as that of the paretic eye. Indeed, the patient is then obliged to lower his head in order to make good the insufficiency of his depressors.

At any rate, we should have recourse to this method only in cases of very slight paresis of the superior oblique.

If, on the contrary, the paresis be pronounced, we prefer to operate on the affected eye only. In fact, we find ourselves here in more favorable

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[1] A. Graefe, loc. cit., p. 82.

[2] Von Graefe, 1864, Klinische Monatsblätter für Augenheilkunde; Alf. Graefe, Handbuch der gesammten Augenheilkunde, vi. 4, S. 84; Mauthner, loc. cit., S. 646; Kries, Archiv für Ophthalmologie, xxiv., 1878, S. 117.

conditions than in paralysis of one of the lateral recti. In this latter case there is no resource, no power acting in the direction of the muscle which is put out of condition for work. When there is a paralysis of one of the depressors or levators, on the contrary, there still remains a muscle which acts in the same direction. Thus, in our example of paralysis of the superior oblique, we still have the inferior rectus, whose power we can reinforce by advancement.

If that did not suffice, we could increase the effect of our operation by the tenotomy of one of the levators of the same eye,—the superior rectus or the inferior oblique. The first of these two operations is very simple. The setting back of the inferior oblique would be impracticable, if we had to detach the tendinous insertion of this muscle from the posterior and external surface of the globe. But one can attack the inferior oblique at its origin near the margin of the superior maxillary bone, external to the orifice of the nasal duct. We described the method of this operation in detail some ten years ago.[1]

Theorists object that, since the inferior rectus, besides lowering the eye, turns it inward and gives it a rotation analogous to that produced by paralysis of the superior oblique, the convergent strabismus as well as the temporal inclination of the eye ought to be accentuated by the advancement of the inferior rectus. Our own experience as well as that of our former assistant, Dr. Eperon, has shown that such is not the case. The obliquity of the image as well as the homonymous diplopia disappears as soon as the difference in height has been corrected, whether this be by means of prisms or with the help of an operation. Moreover, we have called attention to the fact that these two qualities of diplopia are often lacking in paresis of the superior oblique, or manifest themselves only when the gaze is sidewise.

Observation teaches us daily that the eyes have a considerable power of harmonizing their movements after operations performed on their motor apparatus, when binocular vision exists. If it does not exist, the slight degree of lateral deviation which accompanies the paralysis of a levator muscle could not constitute a disfigurement, and the rotation around the antero-posterior axis still less so.

Let us now consider the rather rare case of the *isolated paralysis of the inferior oblique.* The eye is directed downward and inward and its vertical meridian is inclined towards the right. The first thing to do in such a case is to advance strongly the superior rectus muscle of the affected eye. If, at the end of several weeks, there still exists a troublesome vertical diplopia, even when the patient looks downward, a very slight tenotomy of the superior rectus of the healthy eye might be considered.

One would in this way fulfil at the same time my principle of re-establishing binocular vision in the most useful direction (slightly downward),

[1] Landolt, La ténotomie de l'oblique inférieur, Archives d'ophthalmologie, 1885, p. 402.

and that of von Graefe, which gives the healthy eye the same direction as the affected one. Indeed, by the artificial weakening of its superior rectus the right eye is directed downward and to the right, and its vertical meridian is inclined towards the right. Hence it turns round three axes in the same manner as the left eye, whose inferior oblique is paralyzed.

*The isolated paralyses of the superior and inferior recti* are likewise very rare. They are occasionally seen, however. Moreover, these muscles may be involved in a pathological process in their immediate vicinity, or by a traumatism which enfeebles them. The surgical treatment, if it be necessary to have recourse to such, will be the same in any of these cases.

It is true that we no longer find ourselves here in as favorable circumstances as in the case of a paralysis of an oblique muscle. The muscle which combines its action with that of an oblique is one of the recti, whose advancement is easy. The muscles which are associated with the superior and inferior recti,—the inferior and the superior oblique,—on the contrary, can hardly be advanced.

Hence, in the case of paralysis of a superior or an inferior rectus, we shall have recourse to the advancement of the paralyzed muscle itself. This advancement ought to be very thorough, and may be rendered still more efficacious by the resection of the muscle.

If there be *paralysis of an inferior rectus,* it may be necessary to add to the advancement the tenotomy of the superior rectus of the same eye, for strabismus sursum vergens always implies great annoyance to the patient. I should not fear even to have recourse to the detachment of the inferior oblique of that eye rather than to the setting back of the inferior rectus of the sound eye.

It might be otherwise in a case of *paralysis of the superior rectus.* If the left eye be the affected one, this eye is directed downward and slightly outward, and its vertical meridian is somewhat inclined towards the temple.

If, in spite of the advancement, with resection, of the paralyzed muscle, there still persist a slight lowering of the left eye, it would be permissible to produce an analogous lowering of the healthy eye, because this direction is one of the most useful directions of sight.

This lowering of the healthy eye might be obtained by the tenotomy of its superior rectus, by that of its inferior oblique, or by the advancement of its inferior rectus. If the difference in height between the two eyes is but slight, I should not object to the first operation (tenotomy of the superior rectus of the sound eye). If, however, the strabismus left is of several degrees, I should give the preference to the advancement of the inferior rectus.

If all the muscles innervated by the common motor oculi are affected with paresis, there exist, at once, crossed diplopia and difference in height of the two images: divergent and sursum vergens strabismus when the patient looks downward, deorsum vergens when he looks upward.

When the horizontal deviation is very considerable relatively to the

vertical deviation, it often suffices to correct the former in order to see the latter disappear. It is best, in all cases, not to cumulate surgical interventions with the purpose of correcting simultaneously the lateral and vertical deviations. It is preferable to await the result of the horizontal strabotomy before proceeding to an operation in the vertical plane.

The same rule is to be followed when the difference in height is the predominant defect. This defect is then to be corrected according to the principles above explained, and the muscles which act horizontally are to be attacked only when the first operation has not sufficed to remedy the divergent strabismus.

But when the strabismus is considerable, and plainly intermediate, one may attempt the correction in both directions at once.

The necessary operations may be performed on the same eye, or the lateral strabismus may be corrected on one eye and the vertical strabismus on the other eye.

It also happens occasionally that both eyes are affected by muscular paralysis. We cannot enter into the details of all the different kinds of strabismus and motor troubles which may result from this. The principles which have just been set forth will suffice for the determination of the best method of operation in each case.

Without opening the record of my operations, I shall cite two especially complicated cases of this kind.

The first concerns a woman, forty years old, presenting indubitable symptoms of locomotor ataxia. She was subject to *divergent* and *deorsum vergens strabismus*, producing a *vertical diplopia of thirty degrees*, and a *crossed diplopia of thirty degrees*.

I knew very well that the tabetic paralyses often disappear spontaneously, but the patient had waited in vain for years for this fortunate occurrence. The diplopia annoyed her considerably, and she entreated me to rid her of it by any means.

There was no possibility of obtaining this correction by means of prismatic glasses. Hence I decided upon operation, and performed, at the same sitting, the advancement of the internal rectus, with the tenotomy of the external rectus on the right eye, and the advancement of the inferior rectus, with the tenotomy of the superior rectus on the left eye.

During the operation I told my students that it was not probable that these operations would exactly correct the complicated and considerable strabismus of our patient; that, at best, we should expect to have a slight under- or over-correction in one or the other direction; but that we should easily be able to remedy this by means of a further operation, if it should become necessary.

Hence I was very happily surprised when the patient, only a few days later, declared that she no longer saw double when looking straight ahead, or when looking downward. Binocular vision was restored, indeed, so perfectly, and without further operation, that the patient, whom I have

followed for years, could go about, attend to affairs, and do manual labor without the least inconvenience.

A much more complicated and more difficult case, especially from the operative point of view, was the following. Princess T. had undergone an operation, by von Graefe, in her infancy, to correct a convergent strabismus. When she came to consult me, her right eye presented a *divergent strabismus of fifteen degrees* and *strabismus deorsum vergens of thirty degrees.*

It was twenty-five years since the operation had been performed, and the patient could not tell me what had been done. She merely recalled that her eyes had been operated on several times.

Even by a simple inspection, one could see that the internal recti of both eyes had been excessively set back. The caruncles were hidden under the internal angle of the lids, the sclerotic was bare to a great extent, and the nasal excursion of each eye was considerably limited, that of the left more than that of the right. The divergent strabismus as well as the defect of mobility of the eyes was very probably the result of a thread-operation. The operation had given rise not only to a most undesirable over-correction of the convergent strabismus, but even to a difference in height between the two eyes. The operator had tried to remedy this, as was easy to see, by the tenotomy of the right superior rectus, and had thus complicated the over-correction in the horizontal plane by a more considerable, and still more disfiguring, vertical deviation.

The primitive strabismus having dated from the patient's early infancy,—that is to say, from an epoch when binocular vision was not well established,—diplopia did not exist. The right eye was hyperopic by two and one-half dioptrics, and possessed visual acuity of from 0.2 to 0.3. The left eye had 0.6 of normal vision, after the correction of its myopic astigmatism of one dioptry.

I tried to obtain the correction of the divergent strabismus by the advancement of the internal rectus, combined with the tenotomy of the external rectus of the left eye, and the correction of the vertical strabismus by the advancement of the superior rectus, primarily set back, of the right eye, combined with the tenotomy of the inferior rectus of the same eye.

In spite of the powerful effect of these four operations, there still remained a certain degree of divergence and of lowering of the right eye. Several weeks later, I remedied these defects by the advancement of the internal rectus of that eye and the tenotomy of the superior rectus of the left eye.

Let us also cite the observation of a *strabismus sursum vergens* traumatic in origin. This case was operated on very successfully by Dr. Eperon.[1]

A man, forty years old, had received a blow from a cow's horn in the

[1] Eperon, De la correction opératoire des déviations oculaires verticales, etc., Archives d'ophthalmologie, 1889.

superior internal region of the orbit, opposite the pulley of the superior oblique. This muscle was paretic, and a vertical diplopia of twelve degrees, homonymous by one degree, was the result of its enfeeblement. Binocular vision was retained over only a very restricted extent (Fig. 32*a*), commencing about the twentieth degree above the horizontal and going to the upper normal limits.

Eperon performed the advancement of the inferior rectus of the affected eye, and thus obtained, at the end of only two weeks, a field of binocular vision represented by Fig. 32*b*. It will be seen that the fusion of the images of the two eyes is restored over all the useful extent of vision,

FIG. 32*a*.

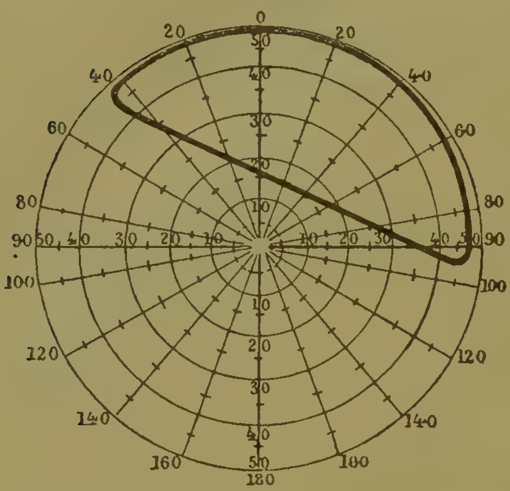
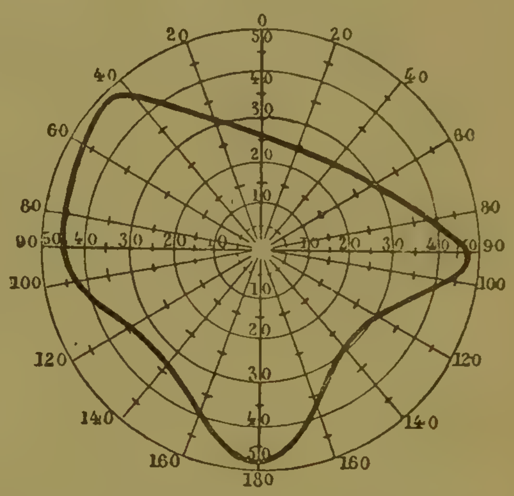


FIG. 32*b*.



—that is to say, for vision downward as well as for that straight forward. Diplopia remained only in the upper right part of the fixation field, which but rarely enters into account. Eight months later, the patient informed the surgeon that he no longer saw double, in whatever direction he looked.

This observation is a striking example of the advantage which muscular advancement has over tenotomy. We shall find additional evidence of the same fact when considering the treatment of non-paralytic strabismus.

When the paralysis of the ocular muscle dates from the earliest years in life, the compensatory position of the head may simulate veritable torticollis.

Cases of this sort, to which I have given the name of *ocular torticollis*, have been reported by Cuignet,[1] Wadsworth,[2] Risley,[3] Morosow,[4] Nieden,[5] Lowett,[6] Bradford,[7] and myself.[8]

[1] Cuignet, Recueil d'ophtalmologie, 1873, p. 24.
[2] Wadsworth, Transactions of the American Ophthalmological Society, 1889, p. 381.
[3] Risley, ibidem.
[4] Morosow, Vrach, 1890, p. 743.
[5] Nieden, Centralblatt für Augenheilkunde, November, 1892.
[6] Lowett, Transactions of the American Orthopedic Association, ii. p. 230.
[7] Bradford, Centralblatt für orthpaedische Chirurgie, etc., 1890, No. 9.
[8] Landolt, Torticollis oculaire, Bulletin médical, 1890, p. 578.

The optical or operative correction of the visual defect is, as a matter of course, at the same time the remedy of this false torticollis, while the application of electricity is of no avail, and operations on the muscles of the neck, which have been proposed by eminent surgeons, would lead to genuine disasters. We cite these cases to show how important it may be to recognize a muscular paralysis and to refer anomalies in the direction of the head to their real cause.

It is also very interesting to note in this connection that, inversely, in true torticollis, the eyes execute a compensating rotation in the opposite direction from that of the face. This rotation, by its persistence, finally brings about a weakening of the muscles which turn the eyes in the pathological direction of the face, and whose paralysis could have brought about a false torticollis.[1]

## II.—NON-PARALYTIC STRABISMUS.[2]

In the foregoing section we have considered the ocular muscles, one by one, with reference to the movement of the globe which the contraction of each produces, when normally exercised, and the phenomena which result from the paralysis of the individual muscle.

Although paralysis frequently affects one single muscle of the eye, it scarcely ever happens that a single muscle contracts separately. All the muscles are always more or less active in giving the eye its direction and in holding it in its position.

A more nearly correct notion of the ocular movements—an idea which is more in conformity with reality—will be had by considering all the muscles as together forming one motor apparatus, in which they combine their action in marvellous harmony. Anatomy, in showing the muscles connected by the capsule of Tenon, the conjunctiva, and the intermediate cellular tissue into one apparatus, makes us comprehend the fusion of their action which gradually goes from one to the other without interruption.

On the other hand, it can be seen that each movement given to the eye encounters an opposite regulating movement. To the forces which draw the globe into the orbit (the four recti), nature opposes, besides the suspensory ligament (the diaphragm of the orbit), the oblique muscles, which tend to draw the globe forward. The abductor muscles, of which the external recti are the principal, have antagonists in the internal recti, the levators are opposed by the depressors, and their actions are again combined to prevent the globe from making a faulty rotation around the antero-posterior axis.

This interaction exists not only for the vertical and lateral movements which are so easily explained by the well-known arrangement of the ocular

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[1] Hübscher, Beiträge zur klinische Chirurgie, 1893, x. S. 299.

[2] See Landolt, Archives d'Ophtalmologie, 1897, p. 74.

muscles, but also for the intermediate movements, however complex they may be. There is no direction that the eye cannot easily take and in which it cannot remain without fatigue.

Hence it is justifiable to assert that there exists an admirable harmony in the action of the motor muscles, since, in spite of their fixed origin and their attachment to the globe, six suffice to vary infinitely the movements of the eye.

The mechanism of the motor apparatus, however, will appear more marvellous still, when the *combined movements of the two eyes* are considered.

The *binocular movements* have for their purpose to direct the two eyes always simultaneously towards the point to which the individual's attention is directed. In this way the image of the fixed point is formed simultaneously on the *fovea centralis* of each eye; the two retinal images are fused in a single one, and there is single binocular vision. From this binocular vision results not only a more vivid visual impression, but, above all, the impression *of the distance of the point looked at, and the sensation of the "relief,"—i.e., of the third dimension.*

It is easy to convince one's self that, so long as the human species has existed, the movements of the two eyes have been associated for the purpose of obtaining this binocular vision. Certain movements which are by no means impossible, such as divergence or the raising or lowering of one eye alone, have remained in a rudimentary condition simply because binocular vision never calls for the former, and only rarely—that is, only during the inclination of the head—for the latter.

Other movements, on the contrary, have become so habitual to the eyes that they are always made by both together, as, for instance, the *raising* and *lowering*. One eye cannot be raised or lowered beyond a certain degree without the other following it. As to the *lateral movements*, if the gaze of the left eye be directed to the firmament, from left to right, from one star to the other, the right eye will follow it faithfully, even if it be covered, and *vice versâ*.

The same is true when the object approaches: the eyes will follow it by *converging*. They follow it likewise when it is withdrawn, by diminishing their convergence up to the parallelism of their lines of sight, when the object has reached infinity.

Hence there has been formed an *intimate association between the muscles acting in the vertical, the levators of the two eyes, and the depressors*, so that similar muscles can scarcely dissociate their contraction.

In the *horizontal* plane, or, to speak more correctly, in the *plane of the orbits*, we have, at first, the association of the *abductors* with the *adductors* in the *lateral movements;* then that of the *adductors* in producing *convergence.*

To these must be added the combined action of the *abductors*, not only in the diminution of convergence and in real divergence, such as is provoked by means of abducting prisms, but also that simultaneous con-

traction of the abductors which is indispensable to counterbalance that of the adductors.

This active abduction has been denied, and it has been sought to explain divergence solely by the relaxation of the adductors. Convergence would be then deprived of an antagonistic function. This is scarcely admissible, in view of the mathematical precision with which convergence is produced and maintained.

Moreover, many of those who deny the existence of active abduction still admit that the position of equilibrium or repose of the eyes is sometimes convergence. It may be asked, then, how the eyes get out of this state of *repose*, if it is not by a simultaneous contraction of the abductors.

It has been urged that, if there exist an active power of divergence, we ought to be able to give to the two eyes a divergence equal to the sum of the rotation towards the temple of which each eye alone is capable,—that is to say, of from ninety to one hundred degrees. Since, "in spite of exercise," we succeed in diverging only a few degrees, some consider the absence of the simultaneous contraction of the abductors as demonstrated. In what, however, consisted that exercise of divergence which should demonstrate anything? It was the application during a very limited time of abducting prisms, to which some oculists have subjected themselves. Are such temporary experiments comparable to the powerful impulse of convergence which has been in action almost constantly, for the sake of binocular vision, during the whole existence of the race? And again, the maximum of convergence is much less than double the nasal rotation of each eye alone; the former is about forty-two degrees and the latter more than one hundred.

Why, one might ask, can the internal recti not turn the globes as far in convergence as in the movements which are called associated? This is probably because it is not necessary. It scarcely happens that one needs to look at objects three centimetres distant. And it is for the same reason—that is to say, because it would be superfluous—that active divergence has not been more developed in our race.

Furthermore, divergence is abolished in paralysis of the external recti muscles, though nothing prevents the interni, in this case, from entire relaxation. Finally, there exists a form of convergent strabismus, or an insufficiency of divergence, among neuropaths, ataxics, or persons enfeebled by debilitating diseases, which finds its explanation in the diminution of the power of divergence and not in a spasm of convergence.[1]

Although we speak of the association of muscles and of groups of muscles of the two eyes, it is of course understood that the muscles themselves are not connected from the one eye to the other, but that this connection is found in the brain; that, if it be a question of the simultaneous contraction of the abductor of one eye and of the adductor of the other, no one ignores the fact that the impulse starts from a common centre of innervation; that if we speak of the simultaneous contraction of the adductors, or of the internal recti, or simply of *convergence*, we admit, in just the same way, the existence of a centre of convergence, as we believe in the existence of a centre of divergence, since we admit that the abductors can contract simultaneously.

One evening I was with Charcot, when a colleague timidly suggested that the existence of a centre of convergence must be admitted. The master contented himself with a shrug of the shoulders by way of reply, and he was perfectly right. Since the function exists, it must have its centre in the brain.

Besides the association of the extrinsic muscles with each other, there exists also a very important connection between the *motor apparatus* and the *accommodative apparatus* of the eyes.

The motor apparatus suffices, it is true, to direct both eyes simultaneously towards the object of fixation, and this object is seen *binocularly*

[1] See, among others, Uhthoff, "Ein Fall von Divergenzlaehmung," etc., Berliner klinische Wochenschrift, 1893, Nr. 11.

when its image is received at the same time by the *fovea centralis* of each eye. But will it be seen distinctly? This is another question. The distinctness of vision is, indeed, not a function of the motor muscles; it depends upon the *distinctness* of the retinal image, and this depends, in turn (apart from the transparency of the refractive media and from the form of the refractive surfaces), upon the *optical adaptation* of the eye.

This adaptation is, as we know, controlled by the ciliary muscle.[1] Contraction of this muscle causes the crystalline lens to become more convex, and thus increases the refraction of the eye. When the muscle relaxes the lens is flattened and the refraction diminishes.

We certainly need not be astonished to find intimate relations between two functions which, however different anatomically and physiologically, are yet always exercised simultaneously, with the aim of securing *binocular* and *distinct* vision.

These relations are most easily explained in this way. The standard eye is *emmetropic*, or nearly so.[2] In a condition of repose—that is to say, when its accommodation is *nil*—it is adapted to infinity. In order to see binocularly an infinitely distant object,—a star, for instance,—the two eyes ought to be directed parallelly: hence in this case convergence is also *nil*.

When the object approaches, the two functions come simultaneously into play,—the convergence in order to direct the two eyes towards the object, the accommodation to adjust their dioptric apparatus to its distance.

The effort necessary to insure *binocular* and *distinct* vision increases alike for the two systems in proportion as the object comes nearer. It is always the same for both in emmetropia,—that is to say, for normal eyes.

The connection between the two functions in question has in consequence become so intimate that, as Donders has shown, one can scarcely be used without the other.[3] An emmetrope who converges makes, at the same time, an effort of accommodation, and when his eyes are made to diverge by means of pri-ms, he can scarcely any longer accommodate. The same is true for accommodation. Let the emmetrope be made to fix a very distant object. Hide the object from his left eye by means of a screen which, however, will permit us to observe this eye. Although not seeing the object which is fixed by the right eye, the left nevertheless remains directed exactly towards the same point as its fellow.

Let a concave glass be placed in front of the right eye; this lens obliges it to make an effort of accommodation in order to see the object distinctly. Of course this right eye will remain immobile, since the point of fixation has not changed its place; but the other eye will be carried towards the

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[1] Landolt, Refraction and Accommodation, translated by Culver, p. 143.

[2] Mathematical exactness does not exist in physiology. There are great errors, but also gross compensations. Biologic phenomena must be considered *grosso modo;* otherwise they will not be understood.

[3] Landolt, Refraction and Accommodation, p. 200.

nose to an extent proportionate with the strength of the glass. This phenomenon is explainable in a very simple manner. The accommodation being always the same in the two eyes, the accommodative effort made by the right eye has provoked not only an analogous effort in the other eye, but also a proportional effort of convergence.

On the contrary, if, while looking at a near object, I cover one of my eyes, this eye will cease to converge so soon as I furnish the other eye with a sufficiently strong *convex* glass, for this glass renders it unnecessary to make an effort of accommodation to see the object distinctly.

Another conclusive experiment in this connection consists in looking through strong abducting prisms at a near object, the vision of which requires at once an effort of convergence and one of accommodation. These glasses, without influencing the accommodation, force upon us the alternative of seeing distinctly,—that is to say, with an effort of accommodation, but with homonymous diplopia, because of the convergence which this effort necessitates and which is counterbalanced by the prisms,—or of seeing singly, by directing the eyes parallelly or nearly so, as required by the prisms, but indistinctly, because of the deficit in accommodation, accommodation being, under those circumstances, no longer stimulated by convergence.[1]

The converse takes place when both eyes are furnished for near vision with convex glasses which suppress the accommodation. In this case, one sees distinctly, but with crossed diplopia, because the relaxation of accommodation brings about parallelism of the eyes; or singly, but indistinctly, because the accommodation which accompanies the convergence towards the near object, by associating itself with the effect of the convex glasses, increases the refraction of the eyes beyond what is required.

The relationship which, in the human species, unites accommodation and convergence, may become so intimate in the case of an individual who enjoys binocular vision that, without being absolutely emmetropic, but having need of accommodation for near vision, he will direct towards the point of fixation even a covered eye, guided with surprising precision by the accommodation which its fellow exerts in order to see distinctly.

Without accommodation, the direction of the excluded eye is most uncertain, since nothing tells the precise location of the point of fixation. This point may be at any distance whatever on the line of sight of the free eye. It is true that, *if the distance of the object is otherwise known*, the normal man, by this means, directs towards the point of fixation, *approximately*, his lines of sight, even though one of his eyes cannot see the point.[2] Thus, when gazing at his finger, he will converge more or less, knowing that the finger is not at an infinite distance, but near him. This convergence, how-

[1] In order to become accustomed to relax one's accommodation,—during ophthalmoscopic examination, for instance,—there is no better means than to di ect one's eyes parallelly to the extent of obtaining a crossed diplopia.

[2] Hansen-Grut, Fifth International Congress of Ophthalmology, New York, 1876.

ever, cannot, of course, have the precision which results from accommodation;[1] but it is of interest, as showing that the imagination, correct or incorrect as to the situation of the object, may influence the reciprocal direction of the eyes.

Knowing now the relations between the movements of the two eyes, as well as the principal factors which preside over their coöperation, we ought to ask ourselves what is the *direction which the eyes assume when in a condition of absolute rest*,—that is to say, when they are abandoned to the equilibrium of their muscles only. As we shall see later, it is not very easy to answer this question, because it is difficult to take away from the individual all the influences which are capable of modifying the direction of his gaze. Inasmuch as even imagination is among these influences, to be sure of absolute repose it is necessary to deprive the person of his consciousness. In this state, in syncope, in narcosis, in death, *divergence* of the eyes is the rule. This is nearly always the case also in double amaurosis. Two blind eyes almost always diverge.

Experiments made on persons who are asleep have likewise demonstrated that, when abandoned to the free play of their muscles, the eyes rarely converge; they oscillate about parallelism, and almost always take a more or less divergent direction.

Thus the eyes normally *diverge* in a state of rest, but *without* an appreciable *difference in height.*

This fact need hardly surprise us. In the first place, the orbits, those cavities in which the eyes develop and move, diverge very appreciably,—from forty-two to forty-four degrees.[2] Let us next recall the disposition of the external recti muscles. They are rolled on the ocular globes as if they had been forcibly obliged to follow them, as convergence developed, under the impulse of binocular vision, the privilege of a superior race.

We must not forget, indeed, that this simultaneous contraction of the adductors is a movement which is not to be met among the lower mammals, and that it is produced in man only in the interest of the vision with both eyes, synergetically with an effort of accommodation, or, as we shall see later, under the influence of a peculiar disturbance of innervation.

We ought not, then, to be astonished if the eyes, abandoned to themselves, assume a somewhat similar direction to that which they had prior to the formation of the species, and diverge more or less.

Whenever both eyes are not simultaneously directed towards the point to which their possessor directs his attention, *strabismus* is present.

Strabismus is, therefore, especially liable to occur when binocular vision

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[1] Landolt, Seventh International Congress of Ophthalmology, Heidelberg, 1888; and Archiv für Ophthalmologie, xxxv. S. 265.

[2] Merkel, Topographische Anatomie, i. S. 237.

does not exist. We have seen, indeed, that in this case it is only the association between accommodation and convergence which can impress a proper direction upon an eye which is not fixing.

If this association is not present, the eyes deviate,—that is to say, they obey other laws than those dictated by the interest of binocular vision. Their direction, as well as their movements, remain, indeed, subject to the associations which we have considered.

Suppose, for instance, an individual one of whose eyes is amaurotic. Binocular vision is impossible. It would seem as if the blind and useless eye ought to be able to point anywhere. This, however, is not the case. In the first place, this eye will not abandon the level of its fellow. Non-paralytic strabismus sursum or deorsum vergens is very rare, and never attains a degree comparable to that which one usually meets with in lateral strabismus.

Hence the eye which does not fix can deviate only in the horizontal direction,—towards the temple or towards the nose. Observation seems to justify this supposition by showing non-paralytic strabismus as almost always *divergent* or *convergent.* Thus, one often finds in text-books the statement that an amaurotic eye turns away from its fellow in diverging, or aids its fellow in the latter's accommodation by converging.

This conception of strabismus is, however, not entirely correct.

For the same reason that one eye never moves alone, so it cannot deviate alone (unless muscular paralysis be present). Hering has very justly pointed out that, even in the special case when the movement of the object viewed seems to require rotation of only one eye, the other takes an active part in the motion. Let the object of fixation approach or recede along the visual line of one of the eyes; though the other eye alone seems obliged to change its direction in following the object, the muscles of the eye which is apparently immobile nevertheless accompany the contraction of those of the moving eye. When the object comes nearer, both internal recti contract as a result of the same convergence-impulse; only, in the case of the eye directed straight forward, the external rectus, by its contraction, prevents the eye from being carried towards the nose and holds it in its position. The opposite takes place when the convergence relaxes in proportion to the recession of the object.[1]

Something analogous happens in that kind of strabismus with which we are at present occupied. There is no divergence of one eye, as in paralysis

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[1] It is known that during convergence the eyes are not only carried inward, but at the same time undergo a slight rotation around the line of fixation. (See, among others, our experiments, Aubert, Physiologische Optik, in Graefe und Saemisch, Handbuch der gesammten Augenheilkunde, ix. S. 660.)

It is easy to observe by means of our dynamometer that, when the object fixed is brought along the visual line closer to one of the eyes, so that only the other eye appears to move, the eye which is apparently immobile executes the same rotation around its antero-posterior axis as its congener. The luminous line of the instrument seems to separate into two equally oblique lines which at the *punctum proximum* of convergence have only one point in common.

of the internal rectus, or convergence of one eye, as in paralysis of the abducens; but there is divergence or convergence between the two eyes in the same degree.

It is evidently always necessary that one of the eyes fix,—that is to say, that it be directed towards the object which attracts the attention of the individual in such a way that the image of this object shall be formed upon the *fovea centralis.* This gives the appearance of a monocular deviation. But in reality we might call non-paralytic strabismus *binocular,* in opposition to the paralytic strabismus with which only the affected eye is concerned.

Hence it would not be proper to say, in a case of strabismus of the *left* eye, that this eye diverges or converges; to express one's self correctly, one would need to say, *the eyes diverge or converge, the right eye performing the fixation.*

The proof that this way of considering non-paralytic strabismus is correct consists in the fact that whichever eye is used for fixation, the excluded eye always deviates *through the same angle.* There is not, as in paralytic strabismus, a *secondary deviation* due to the difference in innervation to which the two eyes are subject.

The angle of non-paralytic strabismus remains also apparently the same during the lateral movements. Hence the term *concomitant strabismus* which is applied to this form of squint.

It has also been called *muscular strabismus.* This name is ill chosen, as we pointed out many years ago.[1] It might, indeed, lead to the supposition that this form of deviation of the eyes was due to changes in the structure of the muscles. If the ocular muscles participate in the evolution of concomitant strabismus, they almost always do it only secondarily.

Strabismus is not always *manifest.* In its early stages, especially, it sometimes remains *latent,* and shows itself only under certain conditions; for example, in consequence of fatigue of the eyes or of the body generally, under the influence of emotion, or, above all, when binocular vision is suppressed by the exclusion of one eye.

Nothing is more instructive than to study the direction which the eyes take under such circumstances. Eyes that are otherwise well directed, by virtue of the power of the impulse to binocular vision, sometimes diverge or converge as soon as the object of fixation is hidden from either of them.

Frequently also, especially at the outset, the deviation is not *permanent.* There exist intervals when the most precise examination reveals no anomaly

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[1] Landolt et Eperon, Mouvements des Yeux, in De Wecker et Landolt, Traité complet, etc., iii. p. 869. See also Krenchel, Archiv für Ophthalmologie, xix. 1873, S. 142; Parinaud, Annales d'Oculistique, 1891 and 1892; Hansen-Grut, Transactions of the International Congress of Medical Sciences, Copenhagen, 1884; Bidrag til Laeren von Skelen, Nordisk ophthalmologisk Tidsskrift, 1889; Transactions of the Ophthalmological Society, x. 1889, p. 1; Helmholtz, Festschrift, 1891, S. 69; Berry, Diseases of the Eye (1st edition), p. 629; Ophthalmic Review, 1893, p. 285.

in the direction of the lines of sight, while at other times there may exist a very evident deviation. In such cases the strabismus is called *intermittent* or *periodic*.

When the vision of each eye is good, strabismus often passes from one eye to the other. Without the fixation-object being hidden from either, sometimes it is one and sometimes the other eye which deviates. This *alternation* of the strabismus might appear surprising; for, while it is quite natural that an amblyopic eye may deviate without producing any visual disturbance, it is not easy to understand how a person whose eyes are not both directed simultaneously towards the object of fixation does not see double.

The *absence of diplopia* is one of the characteristic signs of concomitant strabismus. Yet the statement made in manuals that double vision is never present in this form of squint is not accurate. If pains are taken, one can almost always cause the patient to perceive the image received by his squinting eye simultaneously with that of the other eye. For this purpose a red glass is placed in front of the better eye. The patient is made to look at a distant candle-flame, and the eyes are alternately covered and uncovered for a certain time. Thus, one seeks to cause the patient to perceive separately, then simultaneously, the two differently colored images. If there is difficulty in perceiving one of the images, the screen should be made to pass in rapid succession before the eye usually employed for fixation, in order to diminish the intensity of its vision.

On the other hand, one may seek to provoke diplopia, primarily in the vertical meridian, by means of a prism which carries the image of the object of fixation above or below the horizontal meridian of the retina. If the vision of the deviated eye be at all sufficient, the patient will promptly appreciate the fact that he sees two objects, not only with a vertical interval, but also separated laterally. However, that which distinguishes the diplopia of concomitant strabismus from the diplopia accompanying paralysis of the ocular muscles, is not only the difficulty experienced in provoking it, but still more the *impossibility of determining its exact degree*. Although seeing double, the patient seems to project the two images to the same place; sometimes he can hardly appreciate whether his diplopia is homonymous or crossed. It is as if he were conscious of the false direction of his eyes, and made the correction of it enter into the projection of his retinal images. The victim of paralytic strabismus, on the contrary, projects absolutely as if he did not squint,—that is to say, as if the direction of his eyes corresponded to the normal innervation of their muscles.

Apart from the special conditions which we have just mentioned, the vision of the object of fixation remains single. The image received by the deviating eye seems to be systematically disregarded by the visual centre, which registers only the image received by the healthy eye.

It is easy to represent to one's self this phenomenon, which has its analogue in the domain of physiological facts. Whoever practises micro-

scopy (or ophthalmoscopy) with both eyes open knows that the attention may be concentrated on one visual field to such a degree as entirely to annul the perception of the visual field of the other. He equally knows that voluntarily he can perceive the macular impression of either eye. He may even perceive simultaneously the different images formed on the maculæ of the two eyes. This happens, for instance, in microscopic work, when one eye is used for the examination of the object and the other is employed to sketch it.

Something analogous may be observed in certain cases of strabismus, especially when the deviating eye possesses a fairly good vision. If the image of a well-defined object be produced upon the macula of the deviating eye (especially if one directs the individual's attention to the object), the visual impression of the other eye sometimes fades before that of the squinting eye does. At times also one succeeds in making the patient perceive the two objects simultaneously. Curious to say, when the two objects are similar,—for instance, two candle-flames (one of which may be colored by a glass),—their macular images are not always fused into a single image. The patient passes rather from monocular vision into diplopia.

The phenomenon of "suppression" or of "exclusion" of the images received by the deviating eye has nothing about it, therefore, which ought to be surprising. It is all the more natural since this defect in binocular vision occurs at an age when this function has not yet acquired its entire development, and when it is, therefore, readily susceptible to modifications, as is true of all the other functions of the central nervous system.

Between diplopia as it manifests itself in paralytic strabismus and the absolute suppression of the images received by the deviating eye, exist different intermediate categories, representing so many stages in the process of exclusion which we have just discussed. There are, indeed, persons affected with strabismus in whom lateral diplopia can be provoked only by interposing before one eye a prism in the horizontal direction. The distance between the double images thus received corresponds to the angle of strabismus, increased or diminished by the deviation produced by the prism. The suppression of the visual function occurs, therefore, only for a limited area of the retina, situated between the *fovea centralis* and that part of the retina on which the image of the object of fixation is formed. This phenomenon is designated by the term *regional exclusion.*

In other cases of regional exclusion the localization of the retinal impression of the deviating eye is much more uncertain. The distance between the two images varies, and no longer corresponds to the sum of or the difference between the prismatic and the strabotic deviations.

The region of exclusion may grow larger, beyond the limits above mentioned, and it is always more extended in the horizontal than in the vertical direction. Weak vertical prisms often suffice to provoke a corresponding diplopia, while sometimes, even in the absence of compensating movements of the eyes, very strong horizontal prisms are necessary for the

doubling of the image of the point of fixation in the horizontal direction (*horizontal exclusion*). Again, exclusion may take place over the entire retina; diplopia cannot be obtained by any means. This is the *total exclusion* which actually corresponds to the absence of binocular vision.

In addition to these diverse alterations of vision, we find not rarely that a kind of *new identity between the two retinæ* is established. The part of the retina of the affected eye which receives the image of the object looked at by the normal eye assumes, as it were, the function of the macula. It becomes the centre of orientation for the squinting eye. Its visual impressions may even be fused with those of the macula of the healthy eye. In this case weak prisms suffice to produce diplopia. If, on the other hand, by means of prisms or suitably placed mirrors, we cause the image of an object to fall simultaneously upon the fovea centralis of each eye, instead of fusing the two images, the patient manifests diplopia, and the distance between the two images corresponds to the degree of the strabismus.

The same thing occurs in these cases after successful operation for strabismus. The eyes being now directed normally, instead of binocular fusion of their macular images a marked diplopia—one of the same degree as, but opposite to, that of the former strabismus—is manifest. For instance, if we have by an operation corrected a convergent strabismus of twenty-five degrees, the patient may manifest a crossed diplopia of the same amount. If we have corrected only twenty degrees, leaving five degrees of convergence, the diplopia will be of twenty degrees.

We consider this change in the correspondence of the retinæ of the two eyes as a consequence of the long duration of the strabismus, and not as congenital, as has been alleged by some who have tried to explain by it the deviation. Observation proves, indeed, that after the correction of the squint the diplopia produced by the change in the reciprocal direction of the two eyes almost always disappears.

As to the *sense of depth*, or of the third dimension, in strabotic patients, the investigations which have been made with Hering's experiment show that, in the immense majority of cases, persons affected with concomitant strabismus are incapable of correctly judging where the balls fall.

One sometimes succeeds in giving the strabotic person the impression of "*relief*" by means of the *stereoscope*. For this, however, it is essential that the vision of the deviating eye be not too low, and that the patient be willing to undertake, patiently and methodically, the necessary exercises.

Stereoscopic fusion with impression of the third dimension may in cases of strabismus be so intimately interlinked with a permanent deviation that it is sometimes suppressed by the straightening of the eye. (Nagel, Schoeler.[1]) It may exist without there being binocular fusion in ordinary

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[1] Nagel, Das Sehen mit zwei Augen, 1861; Schoeler, loco citato. See also R. Greeff, Klinische Monatsblätter, 1895, S. 352.

vision, as has been demonstrated by means of Hering's experiment; this is a consequence of the fact, discovered by Schoeler, that the stereoscopic impression may result from the fusion of images formed on regions of the two retinæ, which do not correspond.

In any case, one succeeds with most strabotic persons, when the deviating eye is not too weak, in causing to be perceived simultaneously, or even to be fused, the two components of a stereoscopic image.

Whatever may be the gravity of the changes undergone by binocular vision, the deviating eye is, however, not entirely lost to the patient; it contributes, at least, to extend the field of his vision.

When the deviating eye has sufficient vision and is used temporarily for fixation, it generally projects correctly the retinal images that it receives, the sensorium being conscious of the abnormal direction of this eye.

It happens, on the other hand, in cases of inveterate strabismus, that when the good eye is occluded the squinting eye is very imperfectly or not at all straightened, and that it nevertheless projects retinal images as if it were normally directed. This phenomenon is only the corollary of that which has been mentioned when speaking of the fusion of an eccentric image, received by the strabotic eye, with the macular image of the other eye. The two observations show that an eccentric part of the retina may, in the deviating eye, gradually acquire the functions of the macula and become the centre of orientation for this eye.

Von Graefe, Javal, Nagel, and the author have cited cases wherein the defective eye projected its retinal image simultaneously in *two different ways:* in one way as if it were affected with paralytic strabismus,—that is to say, as if the eye supposed itself to be in the normal position; on the other hand, correctly,—that is to say, as if the part of the retina on which the image falls had taken the place of the macula and corresponded to that of the other eye. It follows that in such cases there would be at once binocular vision and diplopia. Indeed, one of the projections of the deviating eye is confounded with that of the healthy eye, while the other is separately perceived. It sometimes even happens that the three images are perceived each by itself, and that the patient claims to have a genuine triplopia.

We shall recall all these peculiarities of the vision of strabotic persons when we come to discuss the surgical correction of ocular deviations.

## NON-PARALYTIC CONVERGENT STRABISMUS.

*Etiology of Convergent Strabismus.*—The fact of the frequency of convergent strabismus among hyperopes, especially in the medium degrees of hyperopia, is, thanks to the remarkable researches of Donders, known to nearly everybody. That great physiologist explained this fact by the relations between convergence and accommodation. Hyperopes, he says, are obliged to make an effort of accommodation in order to remedy the insufficiency of their static refraction. This effort of accommodation among

those whose *relative amplitude* is not especially developed of necessity entails an excess of convergence.

This phenomenon is still more easily produced when the hyperope looks at a near object the fixation of which in his case requires all the stronger an effort of accommodation. To this greater quota of accommodation corresponds a still more exaggerated amount of convergence. Hence the tendency among hyperopes to a pathological convergence.

It is evident that this excess of convergence cannot manifest itself simultaneously in both eyes; otherwise neither of the lines of sight would be directed towards the fixation-point, unless the head executed a rotation equivalent to that of the fixing eye in the opposite direction. In reality this rotation occurs only in the minority of cases, when it gives rise to a peculiar position of the head. For the most part, the better eye appears straight in relation to the face, whilst the other is turned towards the nose at an angle corresponding to the nervous impulse affecting the adductor muscles of both eyes.

There occurs in this case a phenomenon analogous to that which we have described before, when the object of fixation approaches from infinity along the line of sight of one of the eyes. The innervation necessary to keep the eyes directed upon the object is equally divided between the muscular apparatus of the two globes, but it produces an effect different from symmetrical convergence. In one of the eyes it maintains, so to speak, the equilibrium between the abductor and adductor muscles; in the other it affects especially the adductor, which brings about an angle of convergence double that which would be necessary if the fixation-point were on the median line,—that is, if the convergence were symmetrical. Only in this latter case, in which the nervous impulse distributed to the muscles is exactly proportionate to the distance of the fixation-object, the two lines of sight cross at that object; in the case of convergent strabismus, in which this impulse is exaggerated, the two lines of sight cross *between the eyes and the object.*

An example will make the matter clear. Suppose a young emmetrope to fix an object situated one-fourth of a metre in front of him. He makes an effort of accommodation amounting to 4 D., and a corresponding effort of convergence of four metre-angles for each eye, the object being situated on the median line.[1]

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[1] We have described on page 185 of our treatise on refraction the ingenious method which Nagel has devised to express the *degree of convergence* as well as that of the *refraction* or *accommodation*. Nagel takes as the unit of measurement for both functions the *metre*, and calls the angle of convergence necessary to each eye to fix a point situated at one metre from it on the median line a "*metre-angle.*" The "*metre-lens*," or *dioptry*, represents the refraction required in order to see distinctly this same point at the same distance. In emmetropia this refractive power is furnished by the accommodation.

The effort being, for both functions, greater in proportion as the object is nearer, it may be considered as *inversely proportional to the distance between the object and the eye.*

A point situated at one-half of a metre on the median line will require of each eye, in

In this way he is exactly adapted to the distance of the object, and his two lines of sight intersect at the object.

None of these conditions has changed when the object leaves the median line and is carried directly in front of, say, the right eye. The left eye then furnishes eight metre-angles of convergence, while the right has its line of sight parallel with the median plane. This is not equivalent to saying that the innervation is unequally distributed to the muscles of the two eyes. Only, to the impulse necessary to convergence for one-fourth of a metre is joined the effort necessary for associated movement, which has carried both eyes to the right. Hering shows that the right eye has received the same impulse to convergence as the left. The rotation around the antero-posterior axis is the same for both eyes, though one of them seems to remain in the primary position, while the other presents the appearance of an exaggerated adduction.

If, in the emmetrope, we screen off the fixation-object from the left eye, it still continues to be directed to the same point as its neighbor, even if the position of the object be changed. The reason of this is that in emmetropia the accommodation is equal to the convergence, and therefore both eyes remain directed to the same point even when binocular vision is not present. If now a *concave* glass is placed in front of the right eye, distinct vision can be obtained only by an effort of accommodation. It will then be found that, while the right eye has remained immobile, as the fixation-point has not been changed, the other eye has been turned inward. The stronger the glass, the more is the left eye rotated towards the nose.

Accommodation is always the same in both eyes. Thus, the accommodative effort in the right eye causes not only a similar effort in the left, but also brings about a proportionate degree of convergence. If the concave glass is one of three dioptries, then the effort of accommodation will be three dioptries, and the convergence used will be three metre-angles for each eye. Since, however, it is necessary that one of the eyes—the right in our example—be directed towards the object which is occupying the patient's attention, the convergence will appear to affect only the excluded eye. In the case we are discussing, the left eye will be turned towards the nose six metre-angles while the other looks at a distant object through a concave lens of three dioptries. The same or nearly the same thing occurs in hypermetropia, for hypermetropic eyes may be considered as emmetropic eyes to which concave lenses have been applied.

Let us take the case of a young *hyperope* of three dioptries, and let us suppose that the relation between accommodation and convergence is the same with him as with the emmetrope. In order to accommodate for a distance of one-quarter of a metre, our hyperope brings into play not

order to be seen single, $\frac{1m}{\frac{1}{2}} = 2ma$ of *convergence*, and, in order to be seen *distinctly*, 2 *dioptries of refraction*—that is to say, of *accommodation*—when the eyes are emmetropic. At one-sixth of a metre the convergence will be $6ma$, the accommodation 6 D., etc. See p. 130.

only 4, but $3+4=7$ D. of dynamic refraction. To these 7 D. correspond $7ma$ of convergence. If both eyes executed each $7ma$ of convergence, the two lines of sight would intersect at $\frac{1m}{7}$, or 14 centimetres, that is eleven centimetres nearer the eye than the fixation point, and the object would appear double. But the hyperope will place the object, by preference, directly in front of one of his eyes. He will make over to the other eye all the convergence, which in our example would amount to $6ma$. Thus, while the fixing eye has a normal direction, the other will have a convergence of $6ma$, equal to about eleven degrees.

It is true, as might be expected, that the relation between accommodation and convergence is not an absolutely fixed one; otherwise every ametrope would have to choose between two alternatives: either to see *distinctly* but *double*, because of the defective direction of his eyes, or to see *singly* but *indistinctly*, the normal direction of his eyes excluding perfect optical adaptation.

A certain latitude exists, therefore, as Donders showed, between accommodation and convergence, a latitude to which he has given the name of *relative* accommodation and convergence.[1] The hypermetrope can accommodate more, the myope less, than the emmetrope, with the same degree of convergence. It is for this reason that ametropes are by no means all doomed to strabismus.

This "relative" amplitude of accommodation and convergence, however, has its limits. The hypermetrope of a certain degree may with difficulty succeed in furnishing the excessive amount of accommodation required by his ametropia, while he maintains convergence equal to that of the emmetrope. If the sight of one eye, then, be feeble (either on account of opacity or irregularity of curvature of the refracting surfaces, or of changes in the fundus) to such a degree that the image received by that eye may be easily disregarded, the patient is in danger of a convergent strabismus. In fact, the diplopia which would otherwise have resulted from the abnormal direction of his eyes causes no inconvenience, since the image of the amblyopic eye cannot enter into competition with the clear and distinct image of the other eye which possesses a good visual acuteness and is directed normally towards the object fixed.[2]

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[1] Landolt, Refraction and Accommodation, p. 196.

[2] At the very time of writing these lines we have observed a case which is very conclusive of Donders's theory of the etiology of convergent strabismus.

A girl twelve years old was brought to us by her parents. Her fine-looking and well-directed eyes presented exteriorly and interiorly no perceptible abnormity. One of them, however,—the left,—scarcely sees at all. It is only by chance that this fact has been discovered. Conversation was upon the inferiority of the left hand to the right. The child took part in the discussion, and, already reasoning after the manner of adults who like to generalize, said, "That is not surprising, since the left eye does not see either." Of course, her companions were astonished. The eyes of all were examined. Everybody saw well with the left eye, excepting the child in question and——her father.

As to the father's inability to see with the left eye, nobody was surprised, since one

This "suppression" of the retinal image of a wrongly directed eye, however, may be acquired by perfectly healthy eyes, if the patient does not succeed in dissociating accommodation from convergence in order to gain distinct vision. In such a case the strabismus is, to begin with, alternate, passing from one eye to the other, until it finally permanently involves the eye whose image the patient can the more easily disregard.

On the other hand, the relations between accommodation and convergence may be suddenly disturbed, as, for example, in paresis of accommodation. Here there is no time for the establishment of new relations between the two functions; the extreme nervous impulse required by the accommodation reacts on the convergence, and convergent strabismus may be established.

Donders pointed out and explained this phenomenon of convergent strabismus following a sudden diminution of the amplitude of accommodation. Thus, it is not rare to see the deformity in question appear in consequence of the weakness of accommodation caused by diphtheria, not only in hyperopes whose refraction error is of medium degree, but also in hyperopes of less degree and in emmetropes. A large proportion of the children who are affected by convergent strabismus present a puny appearance which indicates muscular debility, in which the ciliary muscle may participate.

Convergent strabismus being most often the result of hyperopia, one might suppose that its frequency would increase with the degree of the ametropia. This is, however, not the case. The largest contributions to this form of squint are given by hypermetropes of *average* degree. Donders has explained this fact in the following way. If, as we have seen, slight hypermetropes escape strabismus, thanks to the modification in the relation between their convergence and accommodation in the interest of their distinct binocular vision, hypermetropes of high degree do not squint, because even the strongest converging effort would not suffice to call forth the amount of accommodation required by their ametropia and by the position of the object, especially if clear vision has to be maintained for some time. They, therefore, do not attempt to make an effort of convergence which, no matter how exaggerated, could bring them no advantage.

Convergent strabismus tends to diminish as age advances to such an extent that it is rather rare after the age of forty, and often passes off spontaneously. This fact is not surprising, for we know that the static refraction of the eye increases during the first twenty-five years of life;

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of his eyes had always *squinted*, and it is even popularly known that badly directed eyes see badly.

Examination of the refraction revealed the curious fact that the child was *emmetropic*, while her father was strongly *hyperopic*. Though both persons were victims of congenital amblyopia of one eye, it is only in the case of the hyperopic father that this infirmity has occasioned strabismus. It has not been transmitted to the child, whose blind eye has been correctly guided by its emmetropic fellow.

myopia augments, emmetropia frequently changes to myopia, hypermetropia diminishes in degree; it may become emmetropia or even myopia. We do not wonder, then, when we find less convergent squint among adults than among children, who are more frequently hypermetropic. It is true that accommodation becomes more and more difficult with increasing years, while the convergence remains practically the same. One might, therefore, suppose that the assistance required by the former of these functions from the latter would become greater and greater; but the individual apparently accustoms himself to associate a stronger and stronger accommodation quota with the same quota of convergence, especially when the instinct of binocular and single vision is very powerful. Whatever the explanation, it remains certain that there are factors exercising a favorable and controlling influence on the relation between accommodation and convergence, thus tending to prevent the development or favor the disappearance of a convergent squint. These influences grow stronger with each additional year of age. What they depend on we do not yet fully know; they may be connected with the central organ, with the development of the orbit, with the motor apparatus and the eyeball, or with all these factors together.

The hyperope, who squints when looking at objects close at hand, does not necessarily present convergent deviation during distant vision. This fact may be explained as follows. Let us take a hyperope of 3 D., fourteen years old. Assume that he has an accommodation of 15 D., convergence power amounting to 12*ma*.

The following table indicates the respective quotas of convergence and accommodation brought into action when the object of fixation is gradually brought nearer.

| Fixation at | Dioptries of Accommodation. | *ma* of Convergence. | Quota of Accommodation. | Quota of Convergence. | Ratio of Quota of Accommodation to Quota of Convergence. |
|---|---|---|---|---|---|
| Infinity. | 3 | 0 | $\frac{3}{15}$ | 0 | 1 : 0 |
| 1 m. | 4 | 1 | $\frac{4}{15}$ | $\frac{1}{12}$ | 1 : 0.3 |
| $\frac{1}{2}$ m. | 5 | 2 | $\frac{5}{15}$ | $\frac{2}{12}$ | 1 : 0.5 |
| $\frac{1}{3}$ m. | 6 | 3 | $\frac{6}{15}$ | $\frac{3}{12}$ | 1 : 0.6 |
| $\frac{1}{4}$ m. | 7 | 4 | $\frac{7}{15}$ | $\frac{4}{12}$ | 1 : 0.7 |

If one studies the relations between the two series of quotas, one sees that, *for the hyperope, the quota of convergence increases more and more, relatively to that of the accommodation, as the point of fixation approaches.* A similar table for the *emmetrope* would show that this relation remains *constant* (1 : 1.25 in our example), while in *myopia* the quota of convergence *diminishes* relatively to that of the accommodation, under like circumstances.[1]

[1] Landolt et Eperon, loc. cit., p. 885.

Hence there is nothing astonishing in the fact that a hyperope may not squint when looking far away, while one of his eyes will deviate inward during the fixation of near objects, or that a convergent squint increases under the same conditions.

Furthermore, it is probable that the hypermetrope rarely accommodates enough to neutralize all his hyperopia when looking at a distance. Near vision—that is to say, the vision for working—requires greater distinctness, and therefore demands a greater accommodative effort.

In taking equally into consideration not the *absolute* value, but the *quota* of convergence and of accommodation, and in comparing them in emmetropia and in hyperopia at different epochs of life, one finds that for the hypermetrope the effort of convergence to be associated with that of accommodation diminishes with the age. This fact, along with those mentioned above, might help to explain the diminution and disappearance of convergent strabismus with increase of years.

The convergent strabismus of hyperopes, which is the type of convergent strabismus, is, therefore, according to Donders's explanation, a *spastic*, an *active* strabismus. Just as it is seen to increase as accommodation increases during the fixation of near objects, so it diminishes in proportion as accommodation diminishes for distant vision. Hence one might suppose that it would disappear entirely so soon as the ciliary muscle ceased to contract. Those who claim that, in the absence of any fixation-object, when the gaze is said to be vague, the accommodation must be at rest, are astonished to find that convergent strabismus, nevertheless, frequently persists under such circumstances. The explanation of this fact is, however, not difficult. It is indisputable that young hyperopes have the greatest difficulty in ridding themselves of at least a slight degree of accommodation. They accommodate more or less, even when their gaze is not fixed upon any special object. This latent accommodative effort suffices to maintain convergence of the lines of sight.

Genuine repose of the nervous impulses is obtained only by sleep, whether normal or provoked by anæsthetics. Under these conditions young hyperopes stop squinting in the early stages of the affection. (Stellwag.) The influence of mydriatics is no less conclusive as to the etiology of this form of strabismus. When convergent strabismus is not inveterate with hyperopes, it ceases during paralysis of the accommodation. The ingenious theory of the Dutch physiologist is thus once more confirmed.

One is tempted to say that this theory explains only too well the convergent strabismus of hyperopes. Indeed, one may wonder why *all* hyperopes, at least of the category that we have pointed out as peculiarly liable to this infirmity, do not squint; and, on the other hand, why some individuals happen to contract convergent strabismus without being hyperopes.

The tendency to binocular vision which is innate in our species, on the one hand, and the comparatively lax and always changeable relations be-

tween convergence and accommodation, on the other hand, explain sufficiently the immunity of those who escape strabismus.

As to the others,—those who become strabotic,—we must seek the factors which favor the production of strabismus in the circumstances which entail the development or facilitate the sacrifice of binocular vision, or, again, in the factors which directly favor convergence.

Binocular vision is more certainly developed and maintained in proportion as the vision of both eyes is more nearly equal and more nearly perfect. Whenever one eye is very inferior to the other, whether in consequence of *ametropia*, or of *opacities of its dioptric media*,[1] or of *change in its fundus*, or of *congenital amblyopia*, this eye is of but slight importance in the concurrent action of the two; the impulse to binocular vision will never become very powerful, or it may not be developed at all. In such cases the eyes are abandoned to other factors that are capable of influencing their reciprocal direction. Hyperopes are oftenest the victims of convergent strabismus.

This infirmity, however, may exist, even in the absence of the auxiliary causes that have just been pointed out, in the case of eyes whose visual acuity is equal.

When it goes back, as is most frequently the case, to the first years and even to the first months of infancy, one has no reason to be surprised that binocular vision is incapable of maintaining a proper direction of the two eyes. A fortuitous circumstance which may but transiently exclude one of the eyes from vision may suffice, at that period, to teach the hyperopic child to squint.

On the other hand, there have been called in, to explain the production of convergent strabismus among hyperopes as well as among emmetropes and even myopes, circumstances which *facilitate convergence.*

Thus it is that Donders[2] claims an *excessive positive value of the angle alpha* (the angle comprised between the visual line and the axis of the cornea)[3] as facilitating convergent strabismus.

Other authors have admitted a faulty insertion of the *ocular muscles* as being able to bring about strabismus generally, convergent strabismus especially.

Experiments made by Dobrowolsky[4] and myself[5] in 1872, and recently

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[1] Buffon observed that the production of strabismus is favored by opacities of the dioptric media of one eye and, generally, by difference in the visual acuity and refraction of the two eyes. (Histoire Naturelle, Supplément, iii.)

Stellwag has observed films on the cornea in 22 per cent. of two hundred and eighteen patients affected by convergent strabismus, while of three hundred and fifteen hyperopic eyes without leucoma, 8.54 per cent. only manifested a deviation inward.

[2] Donders, International Congress of Ophthalmology, Paris, 1862; and The Anomalies of Accommodation, etc., p. 297.

[3] Landolt, Refraction and Accommodation, translated by Culver, p. 116.

[4] Dobrowolsky, Klinische Monatsblätter für Augenheilkunde, 1872, ix. S. 473.

[5] Landolt, Annali di Ottalmologia, i., 1872.

again by Botto,[1] agree with Donders's idea in this sense, that they show that the angle included between the macular axis and the papillary axis, the angle which I have called Epsilon ($\varepsilon$), is usually greater in hyperopes than in myopes. If, as is probable, the papilla occupies a more constant place relatively to the insertion of the muscles than does the macula, these experiments would show that the macula is situated more temporally (outward) with hyperopes than with myopes.

Instead of speaking merely of the angle alpha, or of the insertion of certain muscles, one could formulate the two apparently dissimilar theories in the following way.[2] Let the visual organ be considered as composed of two hollow spheres, one enclosed by the other. The inner constitutes the nervous coat, the retina; the outer represents the ocular globe properly so called, with the muscular funnel which envelops and moves it. We may call the latter the protective and motor coat.

These two spheres are distinct from an anatomical as well as from a physiological point of view, and embryology shows that they are developed to a certain degree independently of each other. Hence it is permissible to assume that discord may be produced in the relative arrangement of the two systems. If, for instance, the retina has taken, relatively to the protective and motor envelopes, such a position that the fovea centralis is placed somewhat *outward*, the visual lines will converge relatively to the axes of the ocular globes. This location of the fovea centralis, which is especially frequent among hyperopes, would necessarily favor the production of convergent strabismus. The opposite would take place if the retina had undergone in the course of its development such a rotation as would have brought the fovea nearer to the nose. However, investigations concerning the place of the *macula* relatively to the muscular system are still lacking. These considerations are, in fact, meant only to reconcile the two abovementioned theories.

There has also been brought forward, as another accidental cause of strabismus, the length of the *base-line.* An abnormal nearness of the centres of rotation of the two eyes, by favoring the action of the internal recti, ought to favor the production of convergent strabismus; an abnormal greatness of distance between these centres has been assumed to have the opposite effect. (Mannhardt.) Panas, who defends this theory, thinks that the progressive increase in the length of the base-line which results from the gradual development of the ethmoidal sinuses may insensibly change the muscular equilibrium to the profit of the external recti.

One of the circumstances that have been accused not only of favoring convergent strabismus, but even of provoking it from the outset, is the *excessive development of the internal recti relatively to the external recti.*

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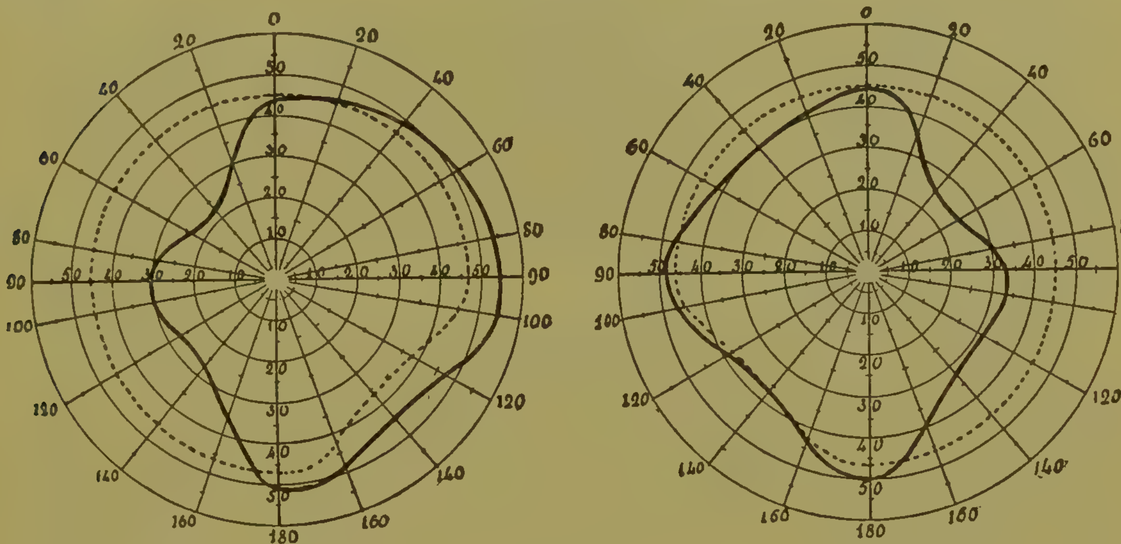
[1] Botto, Thirteenth Congress of the Italian Ophthalmological Society, Palermo, 1892 (Annali di Ottalmologia).

[2] Landolt, International Congress of Ophthalmology, Heidelberg, 1888.

Examination of the field of fixation has taught us[1] the fact, already pointed out by Donders,[2] that the *temporal* excursions of *both* eyes are almost always restricted in convergent strabismus. This defect in motility, which is often not very pronounced in recent cases, is the rule for cases of long standing. It always is found in both eyes, often in the same degree. Frequently it is more developed in the deviating eye than in the fixing one. (Fig. 33.)

FIG. 33.



Fields of fixation of a person affected with a high degree of convergent concomitant strabismus of the left eye.

Moreover, those who, like ourselves, are much given to the performance of muscular advancement have, during the operation, abundant occasion to verify *de visu* the weakness of the external recti muscles in cases of convergent strabismus. These muscles are thin and flabby, especially in comparison with their powerful antagonists.

If the weakness of the external recti, however, is undeniable in convergent strabismus, we have to inquire whether it is primary or secondary. It seems to us that this defective development of the abductors is due to a lack of use. Just as the internal recti become strong by virtue of the constant exercise to which convergence subjects them, so the external recti remain or become weak, since they only very imperfectly counterbalance their antagonists in convergence, and since the associated lateral movements are neither very extensive nor well sustained.

It is, indeed, easy to convince ourselves that in carrying our gaze from one point to another we move our eyes much less than their excursions would permit. If an object, instead of being situated just opposite to us,

[1] The fact, proved by Schneller and by myself, that the limitation of the field of fixation is met with in both eyes, even although the strabismus seems to be localized to one only, proves anew how much reason there is for considering concomitant strabismus as *binocular*, due to an alteration of the convergence, and not as an anomaly in which only one eye is concerned. Schneller, Archiv für Ophthalmologie, 21, 1875, iii. S. 133; and 22, 1876, iv. S. 147; Landolt, Archives d'Ophtalmologie, 1880–81, p. 586.

[2] Donders, Anomalies, etc., p. 303.

is somewhat to one side, or above or below the horizontal, we instinctively have recourse to movements *of the head* to direct our eyes towards it.

I have experimentally determined the excursions which the eyes usually make before we change the direction of our face in order to carry our gaze from one object to another. These excursions do not exceed three or four degrees to the right or left; three degrees in elevation; a little more, say five degrees, in lowering.

From this it will be seen that the associated movements do not make any great demand on the ocular muscles. At all events, no demand is made at all comparable with that made on the internal recti in convergence. We may except the abductor of the fixing eye, which muscle counterbalances the internal rectus when it contracts under the convergence impulse. It is doubtless for this reason that the field of fixation of the eye which is considered the healthy one is often less limited at the temporal side than is that of the deviating eye.

But when, in a case of very pronounced convergent strabismus, there is found a considerable limitation of the temporal excursion of the deviating eye only, without any change in the movements of the other eye, one has almost certainly to do with *paresis* of the external rectus, which paresis dates from the first days of life, and not with a concomitant strabismus.[1]

We have had occasion to mention a limitation of the field of fixation which is quite comparable to that found in strabismus; it is the one Huebscher[2] has discovered in torticollis. The constant rotation of the head in such cases obliges the eyes to be constantly turned in the opposite direction; then the excursions of both of them become limited, by lack of exercise, on the side towards which the face turns, in the same way that their temporal excursion diminishes in the case of convergent strabismus.

Indeed, the *local changes in an ocular muscle* (congenital weakness, vicious insertion, anatomical shortening (A. Graefe), spasm due to a keratitis (Ruete), etc.), which many authors consider the cause of convergent as well as of divergent strabismus, have not been proved to be such, and their importance in the etiology of strabismus has certainly been greatly exaggerated.[3] Hansen-Grut has justly demonstrated that, even if they did exist, they would not produce the symptoms which characterize genuine concomitant strabismus.

It would be wrong, on the other hand, to deny the possibility of such malformations. Why should the ocular muscles be exempt from infirmities which are met with in all the other muscles of the human body? But it is certain that they play only a very secondary part in the production of concomitant strabismus. It is for this reason that in our treatise[4]

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[1] Landolt, International Congress of Ophthalmology, Heidelberg, 1888; and Landolt et Eperon, loc. cit., p. 892.

[2] Huebscher, Beiträge zur klinischen Chirurgie, x., 1893, 2, S. 299.

[3] Vol. x., 1890, p. 1; Gould, The Ophthalmic Record, p. 810, 1894.

[4] Landolt et Eperon in de Wecker et Landolt, Traité complet d'opht., iii., 1887, p. 869.

we protest against the name *muscular strabismus,* which has at times been given to this form of ocular deviation.

Hansen-Grut[1] develops the idea that the *habit* of a certain direction of the eyes may become the cause of strabismus. Thus it is that myopes, who, in accordance with Donders's theory and daily observation, are predisposed to divergent strabismus, may exceptionally acquire a *convergent* strabismus. "Von Graefe," says this author (page 25), "has rightly explained the cause of this form of squint to be the *loss of power in relaxing convergence* induced by continuous convergence." And he adds, "In this case, therefore, it is the continuous habit . . . which ends by not being desisted from." According to Von Graefe, Hansen-Grut, Berry, R. Cross and others, those persons who are constantly engaged with near objects succeed with difficulty in relaxing their internal recti muscles, even during distant vision. At the outset, it is held, this condition is accompanied by homonymous diplopia; but this diplopia is said to be proportionately less troublesome as the eyes are less adapted to the vision of distant objects. Accordingly, it would not be the excessive myopes, who, as we shall soon see, can hardly furnish the convergence required by the nearness of their *punctum remotum,* but the medium myopes, who still enjoy good binocular vision, who should become the victims of this *convergent strabismus* due to habit. However, cases of this sort are very rare, and the ordinary form of strabismus of myopes is divergent strabismus.

According to Hansen-Grut, it is likewise habit which converts the convergent strabismus of hyperopes, from being periodic, into a permanent one. It might be supposed, indeed, that, convergent strabismus of hyperopes being due to an excessive effort of accommodation which is required for distinct vision, this strabismus ought to cease when the accommodation is relaxed. This happens, but only at the beginning of strabismus; later a part of the strabismus remains permanent, even during narcosis. According to Hansen-Grut (*loc. cit.,* page 19), "it is habit, or, in other words, an unconscious innervation, which, owing to frequent excessive convergence (effected in the interest of distinct vision), eventually brings about another functional position of rest than that which originally existed."

Donders said in 1864 (page 303 of his treatise on "Anomalies of Refraction and Accommodation"), "Both the internal muscles of the eyes are therefore to be considered as shortened. The shortening, at first dynamical, as in the case of constant strabismus, becomes organic. It is the result of excessive action with relaxation of the antagonistic muscles."

It is evident that the Dutch physiologist understood by "shortening" of the muscles the diminution in length brought about by their contraction, and he certainly never doubted that this contraction took place under the influence of innervation. Hence the difference between the explanation of

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[1] Hansen-Grut, The Bowman Lecture. Transactions of the Ophthalmological Society of the United Kingdom, 1889–90, pp. 1–41.

permanent strabismus given by the above-named authors, on the one hand, and that given by Donders, on the other, is not very great.

To express the matter simply, we would say: By contracting too much, the internal recti have become unable to relax entirely, or exaggerated convergence finally becomes habitual. Thus, on account of secondary anatomical changes, muscles which at the outset were normal become altered in their functions.

According to Stilling, strabismus is due essentially to the *position of equilibrium* of the eyes; that is to say, to the position which the eyes assume when in a condition of absolute repose. The normal direction is obtained, according to this author, only by virtue of a constant contest against the power of inertia, which pushes the eyes towards their position of minimum innervation. Stilling has found, by means of a method of which we shall have more to say, that this position is rarely parallelism. Generally, the eyes abandoned to themselves diverge or converge. His investigations were made on intelligent persons capable of trustworthy observation, students, doctors, and university professors, none of whom squinted. Now, we question if the result of these experiments justifies the conclusion that it is the position of equilibrium that leads to strabismus. It seems to us rather to indicate that the position of equilibrium does not bring about strabismus, inasmuch as, in spite of the divergence of some and the convergence of others, no one among them had become strabotic.[1]

Moreover, nothing is more difficult than to determine the position of minimum innervation of the ocular muscles. Even, however, if we succeeded in determining for adults this position of equilibrium on which so many authors lay special stress in connection with the genesis of strabismus, would this determination be of very great advantage? We think not. This position of muscular equilibrium is far from being an absolute, an independent, a primary matter. It is, on the contrary, the *consequence* of the influence of a number of factors which act upon the relative direction of the eyes. It is these diverse factors which, in the majority, conduce to the harmony of the movements of the eyes, while they meet, in other persons, with obstacles which impede the normal development of these movements, leave imperfections, insufficiency, or occasion strabismus. Or is one to believe that the position of equilibrium that is found in a man twenty years old is the same that he had when he came into the world, and which predisposed him either to the normal position of the eyes or to strabismus? Have the position and the direction of the eyes not been formed and modified under the influence of the refraction (which, due essentially to the length of the globe, is much more constant than the motor system of the eyes) and of the accommodation, the vision, the occu-

[1] Stilling, Archiv für Augenheilkunde, xv., 1885. See also, among others, Wahlfors, Vom Schielen, etc., Archiv für Augenheilkunde, 1893, S. 207.

pations, and by other circumstances of which we are yet ignorant, at least in part?

All that the position of equilibrium can indicate to us is the relative direction which has become easiest to the eyes by virtue of these various influences,—a tendency to diverge or to converge; the direction in which an eye would probably deviate if binocular vision were to be lost. This position of equilibrium, however, is not the *cause* of strabismus; it can, at the very most, under certain circumstances, determine its character.

Moreover, the fact brought out by Stilling, that in the immense majority of cases ("*in ueberwiegender Mehrzahl der Fälle*") *hyperopes converge* in a state of repose, while myopes in similar circumstances *diverge*, seems very significant. *Emmetropes* occupy the middle place between the two. This result is not at all astonishing. The relation between accommodation and convergence foretold it. Such relations predispose hyperopes to converge, myopes, as we shall see, to diverge.

It may be objected that it is not the position of equilibrium which the eyes present at the age when the Strassburg professor determined it, but that which they had at the beginning of life, which becomes the cause of strabismus. Were this so, his theory would accord with what we have said above concerning a want of harmony between the motor and the visual shells of the eyes. For the equilibrium in which the muscles maintain the ocular globes has always reference to the direction of their visual lines,—that is to say, to the place occupied by the macula relatively to the motor apparatus.

The inanity of the muscular theory of squint having been shown long ago, one hears it said upon different sides that it has been replaced by a new theory, the "central" or "nervous" theory. Some authors seem to believe that this theory differs entirely from that of Donders.

The words "nervous" and "central" have, however, no significance in themselves. When Donders showed that the explanation of squint was to be found in the intimate relations of convergence and accommodation to each other, when, before, with, and after him, clinical workers insisted upon the importance of binocular vision in the etiology of squint, strabismus ceased to be considered a muscular affection, and its causes were referred to the central nervous system, where are to be found the connections between the innervation of convergence and that of accommodation, and where takes place the fusion of the two macular impressions in the sensation of binocular vision.

Donders's theory which we have exposed is, therefore, not only a so-called "nervous" or "central" theory, but it has the great merit of explaining the various phenomena that accompany strabismus, in conformity with daily observation.

Since Donders's day much progress has been made in our knowledge of the anatomy and physiology of the brain, and one may now express in a new language certain things that had by no means escaped the notice

of earlier observers, but which they expressed in terms different from those which we actually use. Thus, for the term "binocular vision," M. Parinaud[1] proposes that of "macular reflex of convergence," this reflex being comparable to the accommodative reflex of convergence, etc.

In the light of more recent cerebral physiology, the etiology of squint will no doubt become clearer. What must not be lost sight of, however, is that the origin of squint is, nevertheless, as before, attributed to the relations between accommodation and convergence and to the alterations of binocular vision. The attempts that are made, reasonably enough, to progress towards a more exact localization of the causes of strabismus in the central organ do not mean that from this date it has to be considered as a properly so-called cerebral affection. Changes in the centres of innervation as *primary* causes of strabismus are admissible only in certain definite cases, and are accompanied by other characteristic symptoms. It is found, for instance, as the consequence of neoplasms, tabes, insular sclerosis, hysteria, neurasthenia, syphilis, morbus Basedowii, chronic alcoholism, etc. Here we are not dealing with strabismus due to lesions of the brain or nervous system. To do so would be to encroach on the domain of our colleagues, Standish, Stevens, and Swanzy, to whom has been assigned the interesting subject of the relations between the visual organ and central affections.[2]

*Essential paralysis of divergence*, which causes convergent strabismus, is often betokened by homonymous diplopia, which persists without notable modification in whatever direction the patient may look.[3] It may even be combined with a defect of convergence[4] and with paralysis of some isolated muscles of the eyes.

Convergent strabismus may be due also to a *spasm of convergence*, independently of accommodation and refraction.[5] We have observed cases of this kind in hysteria.[6] It is perfectly admissible that the same phenomenon is produced in consequence of other irritations of the centre of convergence.

To conclude, however, that *all* strabismus has a similar cause seems to

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[1] Annales d'Oculistique, 1896, p. 401.

[2] Consult on this subject George T. Stevens, Oculo-Muscular Defects and Nervous Troubles, 1880; Oculo-Neural Reflex Irritation, International Medical Congress, 1889; an essay presented to the Royal Academy of Medicine, Brussels, 1884; Parinaud, Paralysies des Mouvements associés des Yeux, Archives de Neurologie, 1882; Noyes On the Tests for Muscular Asthenopia and on Insufficiency of the External Recti Muscles, Transactions of the Eighth International Medical Congress, Copenhagen, 1884; Diseases of the Eye, p. 200; Landolt et Eperon, loco citato, p. 922.

[3] Parinaud, loco citato.

[4] Landolt et Eperon, loco citato, p. 902.

[5] Parinaud, Gazette hebdomadaire, 1879, and Compte-rendu de la Société d'Ophthalmologie, Paris, 1889.

[6] See, among others, Borel, Arch. d'Opht., 1886 and 1887, January, July, August. Hystero-traumatismes oculaires, Congress at Rome, 1894; de Lapersonne, Recueil d'Ophtalmologie.

us to go too far; and to declare that convergent strabismus is due to an excess of energy of the centre for convergence, and *vice versa* for divergent strabismus, is arguing too simply. If one squints towards the nose, without being the victim of paralysis of the abductors, it is because his adductors are not held in equilibrium by the abductors,—because the former contract too much, the latter too little; and since the muscles contract only under the influence of a nervous impulse, it is evident that in this case there must be an absolute or relative excess of the innervation for convergence. Hence this way of explaining strabismus is in reality only a circumlocution. We would like to know *why* the centre of convergence shows itself so exuberant in convergent strabismus, and why in divergent strabismus this centre fails to do its duty.

Donders has first answered this question in a way which is satisfactory for the immense majority of cases of strabismus. He has given an *explanation* of its principal forms, in demonstrating the influence exercised by accommodation and binocular vision upon the direction of the eyes.[1]

The commonest form of convergent strabismus, that which accompanies hyperopia, dates most often from the first years of life.

The occasional cause of the development of deviation often remains obscure. It not infrequently appears after a prolonged exclusion of one eye, as by a bandage used for keratitis *e.g.* In this case, it is evidently the suppression of binocular vision which allows the hyperopic child to facilitate his accommodation by an excess of convergence without being troubled by diplopia. The efforts of near fixation are especially favorable to the development of convergent strabismus. This form of squint usually appears at the time when the child begins to fix his attention upon objects which are shown to him, especially those close at hand, which it can seize, or later, when the study of the alphabet commences to impose its exigencies upon vision. It affects especially young children of puny constitution who have weak accommodation. A certain number of them discover accidentally the artifice of procuring greater distinctness of vision by the sacrifice of one eye. The exaggerated impulse of convergence is then entirely of a reflex nature, as are the movements of our eyes which follow the displacements of our attention in space. Some hyperopic children, on the contrary, as Mauthner points out, learn from their comrades, by imitation, the art of squinting. They squint at first at will, then involuntarily, if not unconsciously. Schweigger cites, conversely, an instance of a young boy who *voluntarily* cured himself of convergent strabismus, and I have published (*Arch. d'Opht.*, xvi. p. 404) the case of a young woman who noticed, while looking at herself in a mirror, that when she saw distinctly with one eye, the other was turned inward. She thus practised squinting or not squinting at will. In the latter case her vision was, of course, indistinct.

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[1] Landolt, L'Etiologie du Strabisme, Arch. d'Opht., 1897, p. 74.

If, as is sometimes the case, the strabismus has developed under the influence of convulsions, we may infer that alterations in the nervous centres have contributed to its production.

Hyperopic convergent strabismus generally commences by being *periodic* or *alternating*. It is oftenest, at the outset, also *relative*. From a very early stage binocular vision has ordinarily undergone profound but still reparable injury. Later the strabismus becomes *permanent*, *absolute*, and localizes itself in one eye, generally in the weaker, though it may remain alternating for years. When it tends to spontaneous cure, it traverses the same stage in the opposite sense; after having been permanent, it resumes its periodicity or relativity, and sometimes may disappear entirely. But, although in such cases the deviation is effaced, complete binocular vision is not always spontaneously re-established.

### TREATMENT OF CONVERGENT STRABISMUS.[1]

The principles of the treatment of convergent strabismus are the logical outcome of what we have just shown concerning its etiology.

The excess of convergence which constitutes this infirmity being in the immense majority of cases due to a defect of refraction which necessitates an exaggerated effort of accommodation, one will try, in the first place, to remedy this defect, whether due to hyperopia or weakness of accommodation, by means of *convex glasses*.[2]

If the spasm of convergence shows itself too tenacious, one must have recourse to the complete suppression of this function by means of *mydriatics*.[3] The accommodation being excluded, convergence is deprived of a very important stimulus.

Moreover, as the production of strabismus is favored by the inferiority of one eye, we must correct the optical errors of the deviated eye, especially astigmatism, and restore its visual power by exercising it.

Still more important than the training of one eye by itself are the exercises which bring into action both eyes simultaneously for the purpose of *stimulating or re-establishing binocular vision.*

The principal cause of strabismus and the greatest obstacle to its cure being the absence of binocular vision, it is evident that anything which tends to re-establish the fusion of the retinal impressions of the two eyes constitutes a valuable therapeutic agent in the treatment of this infirmity.

Finally, if these means, which we shall call *pacific*, are inadequate, we may resort to operative interference.

These are the remedies at our disposal with which to combat convergent strabismus. Let us see now how they may best be used.

It is evident that, if we are dealing with a very young and frolicsome child, we shall not think of making him wear glasses to cure the hyperopia

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[1] Compare Landolt, Refraction and Accommodation, translated by Culver, p. 399.

[2] Donders, Anomalies, p. 305.

[3] John Green, Transactions of the American Ophthalmological Society, 1870, p. 138.

which the ophthalmoscope has revealed. We content ourselves in such cases with the use of mydriatics, taking pains to advise the parents to prevent the child, as much as possible, from looking at small objects near by. Nothing is lost in such cases by deferring energetic treatment. The muscular conditions, the relations between convergence and accommodation, etc., are much modified, and often very advantageously, as the child's development proceeds.

The spontaneous cures, also, of convergent strabismus[1] moderate the medical, and still more the surgical, impatience of the specialist.[2]

If the strabismus affects children of more advanced age, but is still only at its *commencement*,—that is to say, if the exaggeration of convergence has not yet become permanent, and arises only under the influence of a transient act of fixation,—in such cases *resting the eyes*, the cessation of all work necessitating an excessive effort of accommodation, will sometimes suffice to dispel the deviation which otherwise would become persistent.

Nevertheless, in order not to condemn the child to absolute inactivity, especially if it has already begun its studies, we shall relieve the accommodation of at least a part of its work by means of *convex glasses*. Thus it may be that wearing glasses which correct the manifest hyperopia prevents the deviation in young hyperopes, or suffices to cure it when it has just commenced.

It is clear that if hyperopia is accompanied by astigmatism, the latter should be corrected at the same time. In this way the ciliary muscle will be doubly relieved,—first, by being spared the correction of a part of the optical defect; and again, because the patient is thus enabled to hold his work at a greater distance, thanks to the increase of the visual acuteness afforded by the cylindrical glasses. It is best, indeed, in all cases of convergent strabismus to advise the patients to hold the objects of fixation as far away as possible.

According as strabismus is more or less tenacious, one will prescribe the glasses to be worn constantly or only for vision near at hand.

If the strabismus has become *permanent*, even the constant use of glasses which correct the manifest hyperopia will not suffice to dispel the deviation. One would naturally be led to think, in such a case, of excluding even a greater amount of accommodation by correcting the total hyperopia. Such a conclusion would be perfectly logical. But if, for such a patient, we simply prescribe glasses correcting his total hyperopia, we shall never obtain the desired effect, and that for the simple reason that the patient is not able to relax his accommodation sufficiently. In order to achieve our object we must, therefore, have recourse to MYDRIATICS.

Indeed, as soon as we have to deal with a young hyperope whose strabismus has become permanent, we subject him, first of all, to a course of

[1] Von Wecker, Klin. Monatsbl., p. 453, 1871; Schneller, Arch. f. Ophth., xxviii. 3, p. 97, 1882.

[2] Landolt, International Congress of Ophthalmology, Heidelberg, 1888.

treatment with atropine. We instil into both eyes once or twice daily, according to the age of the patient, one drop of a solution of the drug (1 : 400, or 1 : 200). It is advisable to keep the eyes closed for five minutes after the instillation, in order to increase the effect of the mydriatic and to prevent poisoning. If the age of the patient permits, we immediately give him glasses which correct the total hyperopia. In anisometropia each eye will, therefore, be fitted with its correcting lens. For protection against bright light we select smoked and large glasses.

Very young children, for whom the wearing of glasses is impracticable, must be protected from the dazzling light by a veil or shade. In no case do we allow the patient, at the beginning of the treatment, to use his eyes for looking at near objects. We try, indeed, to avoid convergence not only by forbidding the patient to make any attempt at near vision, but also by taking away distinct vision for near objects, and consequently, as much as possible, the inducement to near vision.

If, after a few weeks' properly continued treatment, it appears that, with the required glasses, neither the strabismus for distance nor that for near vision returns, we may order for children of studious habits spectacles to be used while working; but we must at the same time emphatically advise them not to abuse our permission to return to work, and to take it up at first for but a few hours each day, and with frequent intermissions.

Thus it is often possible to continue a course of atropine treatment for months without serious detriment to the child's studies. By and by we may try to diminish the dose of the mydriatic, or even to discontinue it altogether. If this succeeds without the patient again relapsing into squint, we change the glasses which have hitherto been worn, for the strongest convex lenses with which he is able to see at a distance. In anisometropia and astigmatism we follow the rules given elsewhere.[1] The patient must now wear these correcting glasses constantly; but he no longer requires special glasses for working, as his accommodation has again attained its full power.

The strabismus is, in fact, cured, and we may congratulate ourselves upon the result obtained, in spite of the necessity of wearing working glasses. If, after some time has expired, there has been no recurrence of strabismus, we lessen the strength of the convex glasses and endeavor to do without them, at first for seeing at a distance, later at work. In favorable cases we succeed thus in dispensing, little by little, with the use of spectacles. This is the case especially when the hyperopia is of moderate degree, the squint but slight, the visual acuteness good in both eyes, and the constitution robust. Such cases, which are not very rare, constitute real triumphs of the healing art.

Greater difficulties have to be surmounted in the treatment of convergent strabismus where the visual acuteness of one eye is very slight. As

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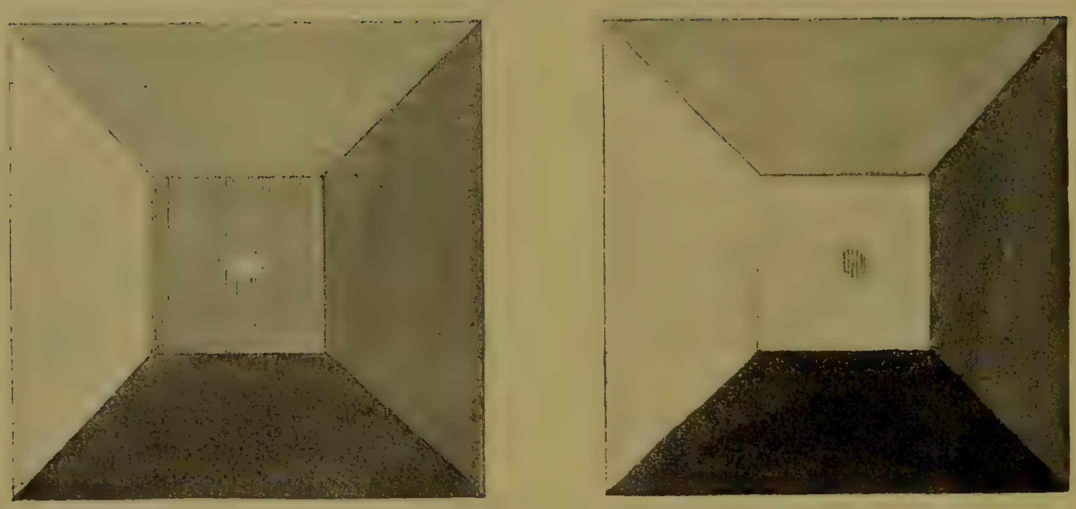
[1] Landolt, Refraction and Accommodation, translated by Culver, p. 526.

Fig. 34.



Dissimilar figures of truncated pyramid.

has been said, and as can be readily understood, it is always the more defective eye which deviates. Besides the course of treatment above indicated, we seek in such cases to restore the weak and deviating eye to the accomplishment of its normal functions. Therefore, errors of refraction will need to be most carefully corrected, and we must seek to augment its visual power by special training. Such training must, however, always be undergone while the accommodation of both eyes is excluded, and must, moreover, never be continued until fatigue sets in; otherwise we incur the risk of increasing the strabismus. The squint naturally manifests itself in the healthy eye as long as the weak one fixes. This is the reason why in such cases we always add to the training of each eye by itself stereoscopic exercises involving the simultaneous use of both eyes.

In this way may be achieved an infinitely more important result than the improvement of the visual acuity of the deviating eye,—namely, the re-establishment and consolidation of *binocular vision*, our most powerful aid in every therapeutic attempt to combat strabismus.

Although Dubois-Reymond had already foreseen the service that the stereoscope might render in the cure of strabismus, it is to Javal[1] that we owe the principles and the development of its use in ophthalmology.

The principle of the stereoscope exercise is to present to the eyes two images, differing from each other in such a way that, by their fusion into one, the impression of a solid, of perspective, of the third dimension, may result.

To begin with, two vertical lines may be used, one above, the other below the horizontal, which, seen stereoscopically, become one single line. (Fig. 41.)

In order to facilitate the fusion of the stereoscopic images they should have some parts in common. If the images are letters, the words which they compose thus become complete only by the fusion of the two parts. Those marks are likewise to be found in the very practical stereoscopic figures devised by Dr. Fromont,[2] chief surgeon of the Belgian army, in those of Dr. Dahlfeld, and in the more recent ones of Dr. Javal.

In our Fig. 34 the central point which is common to the two drawings will guide the patient's eyes. Another mark is formed by the striped square, in which, in the figure on the left, the central circle is lacking, while it is found on the right, where the rest of the square is empty. The fusion of the two slightly dissimilar figures of a truncated pyramid will give to the individual a vivid sensation of perspective.

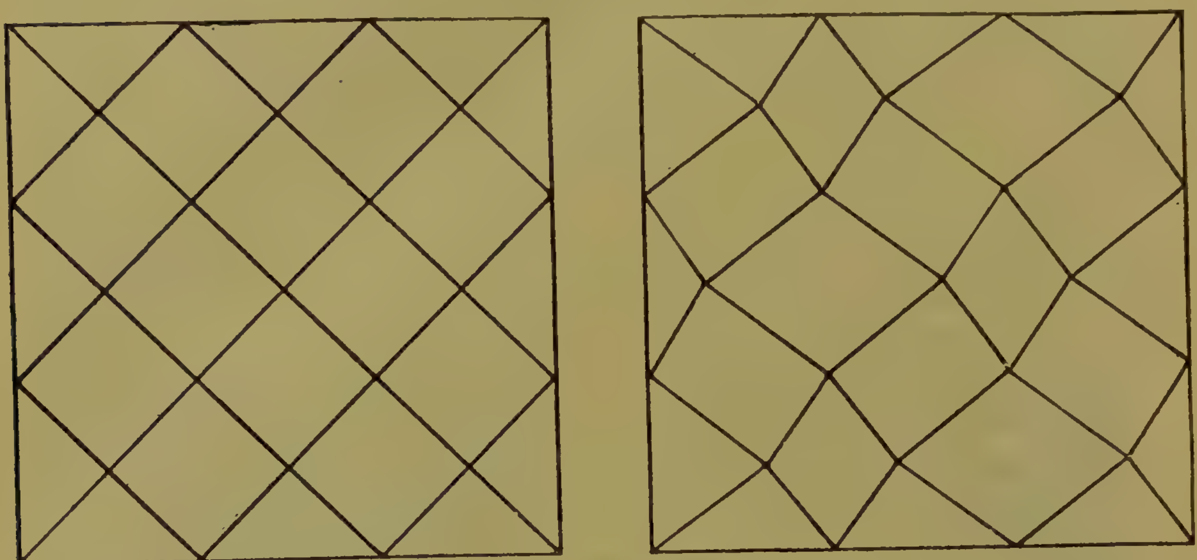
We always take care, when requiring a patient to look at stereoscopic figures, to furnish each one of them with points which alone can surely tell us if the patient is taking account of the perspective solely by the aspect of the picture itself or by stereoscopic fusion of the two. Thus,

[1] Javal, Ann. d'Oc., lxv. pp. 197, 971; lvi. pp. 5, 113, 209. Bulletin de l'Acad. de Médecine, xvi. p. 803, v. Manuel du Strabisme, 1896.

[2] International Congress of the Medical Sciences, Brussels, 1875.

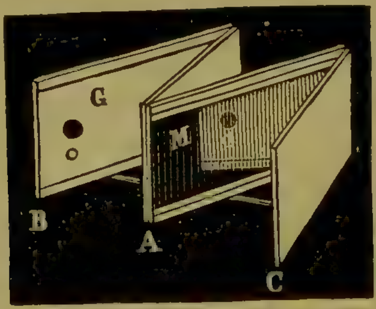
it will be noticed in Fig. 35, which represents an avenue in the park at Versailles, that in the left picture the statue on the right of the foreground bears a black spot, while in the right picture it is the statue on the left which bears the black spot. In the fusion picture both statues ought, necessarily, to be marked. If the patient says that he sees only one which is marked, and tells which one it is, we know at once that he is using but

Fig. 36.



one eye, and we know which eye he employs. There may be noticed also the spot on the reflection of the right statue in the right figure. This enables us to ask the patient whether or not the reflections from the water are like the corresponding statues. At times we also use two points on one side and one point on the other, as in the sky in Fig. 35. We place

Fig. 37.




Wheatstone's mirror-stereoscope.

them so that in the fusion picture the three points will be on the same line, one between the others.

By the aid of these marks an attentive patient may exercise quite by himself, watching as to whether he sees monocularly or binocularly.[1]

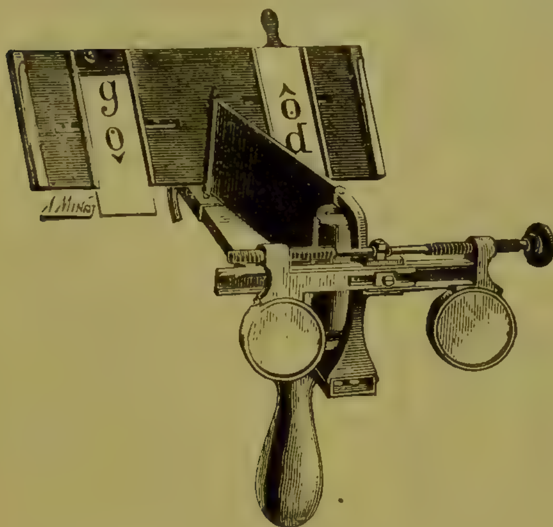
[1] As regards the photographic views which one finds in commerce, it is to be noticed that the corresponding points of the two pictures are much farther apart than are the eyes. Hence one nearly always needs to have recourse to adducting prisms to facilitate fusion, or to cut off a piece of each, in order to use them in our stereoscope without prisms.

FIG. 35.

Corresponding points on fusion pictures.

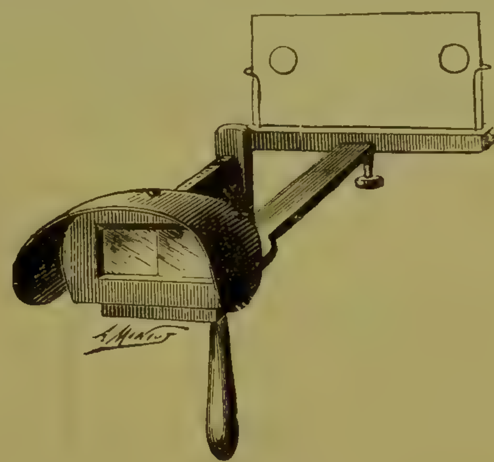
The most useful pictures are those which, like Wheatstone's (Fig. 36), procure, for the person who sees them binocularly, the impression of a solid.

FIG. 38.



Javal's stereoscope with five movements.

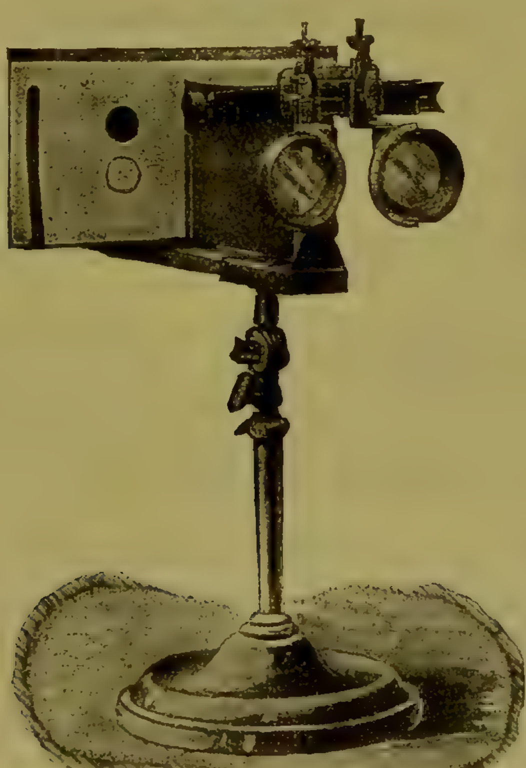
FIG. 39.



Holmes's stereoscope.

Any stereoscope may be used for these exercises. Javal formerly used Wheatstone's *stereoscope with mirrors* (Fig. 37). Since then he has per-

FIG. 40.



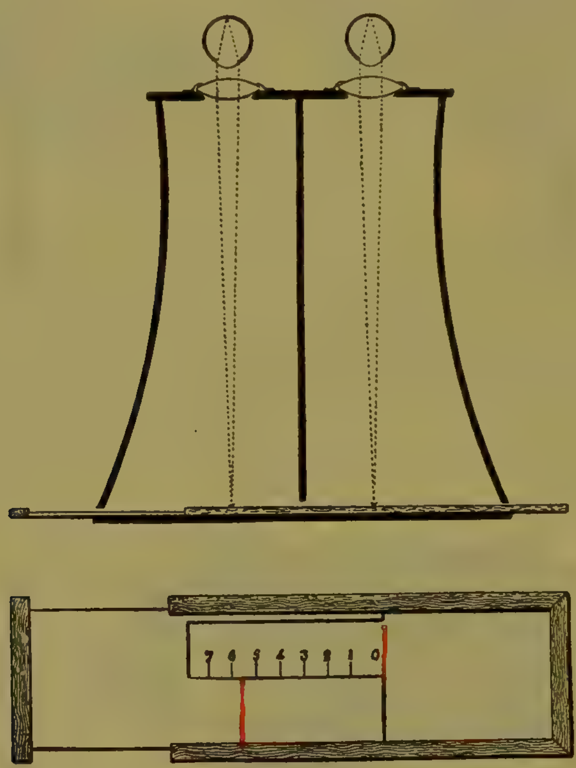
Oliver's stereoscope.

fected his method by the five movements' stereoscope (Fig. 38). This instrument may be regarded as a derivative from the well-known stereoscope of Holmes (Fig. 39), the Mexican or American stereoscope. It is susceptible of more varied applications than the latter.

Another stereoscope very useful for our purpose is that devised by Oliver (Fig. 40). It is susceptible of eleven different forms of adjustment.

We employ for the re-establishment of binocular vision a very simple apparatus[1] (Fig. 41). It consists of an ordinary stereoscope box, from which we have removed the prisms, in order to substitute for them whatever glasses from the trial-case may be desirable, whether spheric, cylindric, prismatic, or a combination of them.

FIG. 41.




Landolt's stereoscope.

The upper part of the figure represents the stereoscope box furnished with glasses which adapt each of the two eyes to the distance of the visual objects.

These latter are fixed to two boards which slide one upon the other in such a way that one can alter at will the distance between the two figures. This distance is indicated in millimetres by the division of one of the boards.

The figures to be fused are represented here by two simple red lines, the one corresponding to the point zero, the other to the sixth centimetre of the division.

It is well to make the partition of the stereoscope rather long, in order to prevent one eye from trespassing on the visual field of the other. The visual objects placed at the opposite end of the box from the eyes to be exercised may be brought nearer to or separated from each other at will. They may even be raised one above the other, or inclined relatively to each other.

The principle of any stereoscope useful for the treatment of strabismus is that the instrument employed *places the eyes in the conditions which are most favorable to the fusion of their retinal images.*

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[1] Landolt, article Strabisme, in Dict. Encyclopédique des Sciences Médicales, Paris, 1883; and Refraction and Accommodation of the Eye, translated by Culver, p. 408.

One of the eyes being generally weaker, or in any case less exercised, than the other, it is a question, first of all, of making its visual impressions as vivid as possible. For that purpose we correct its astigmatism and adapt it with perfect precision to the distance of the object which it is to see, in order to procure for it an absolutely clear retinal image. After doing so, we must seek the relative position of the two figures in which their fusion is possible.

Let us take, for example, our stereoscope and place the test objects at a distance from each other approximately equal to that between the two eyes. Under such circumstances their fusion into one single impression necessitates the parallel direction of the eyes. Knowing that this parallelism is generally possible only in the absence of any accommodative impulse, we provide the patient's eyes, or the sight-holes of our stereoscope, with glasses which permit him to see at the distance of the objects without any effort of accommodation.

Ordinary stereoscopes being generally about 166 millimetres ($\frac{1}{6}$ metre) deep, emmetropic eyes would require convex 6 D. to fulfil this condition. If we have to do with a hyperope of 4 D., we shall give him convex 10 (4 D. to correct his hyperopia and 6 D. more to adapt him for 166 millimetres).

It will be noticed, however, that, even under such circumstances, the majority of patients do not succeed in fusing the images. This may be due merely to incapacity to direct their eyes parallelly. We then help the patient to find the distance between the objects which is requisite for the fusion of their images. When he has succeeded in doing this, we shall gradually separate the objects more and more until fusion is effected with perfect parallelism, or even slight divergence of the lines of fixation.

But the greater part of the time the patients do not fuse, whatever may be the interval between the two objects. They see alternately the one or the other, when the vision of the one eye is about as good as that of the other, or only the object corresponding to the better eye when the vision of the two eyes is different.

In this case it becomes a question of attracting the attention of the weaker eye to the object which corresponds to it, to cause it to fix energetically, excluding its congener. By successively covering and uncovering the better eye for longer or shorter intervals,—indeed, even by furnishing it with a too strong convex glass which lessens the clearness of its vision,—one may succeed in causing the patient to see the two objects simultaneously, and at length to fuse them into a single one. Much patience is required, in order to achieve this result, on the part of the surgeon as well as of the patient. It is for this reason that this training, so logical and useful, is far from having found in ophthalmic practice the place which it deserves.

There exists, indeed, an infinity of forms—sometimes very strange ones—of incapacity of fusion. There are persons, for instance, who succeed

in seeing double,—that is to say, in perceiving simultaneously the visual impression of the two eyes. It might be supposed that nothing could be easier than, by means of prisms or of the stereoscope, to cause the retinal images to fall simultaneously on the *fovea centralis* of each eye, and thus to obtain the fusion of the two. This is, however, often impossible. Just as in those cases homonymous diplopia changes suddenly into crossed diplopia as soon as the double images have been brought very near each other, similarly in the stereoscope the images change sides rather than become fused. It is for such cases that Javal has devised as test object for one of the eyes a wafer surmounted by a black point, while that for the other eye is a series of such wafers, so placed that the fusion of the former with one of the latter becomes almost inevitable. The little point serves as a mark by which to know if fusion has taken place, and with what amount of separation of the test objects. But such is the alteration of binocular vision in some cases, that even this experiment, which would seem to be absolutely conclusive, is not entirely so. The author of this method has proved that sometimes the deviating eye disregards the image received by its *fovea centralis* (the wafer), and only perceives that of the little point which is formed on an eccentric part of its retina.

When the patient is able to see simultaneously both the images in the stereoscope, this is certainly already a satisfactory result. The goal, however, is not yet reached. Binocular vision worthy of the name cannot be considered attained until the patient is fully conscious of the third dimension, a sensation that results only from the fusion of the two different views of the same object. That is the reason why we find the exercises with that sort of figures so useful.

The best test of binocular vision is the well-known experiment of Hering. We rely upon it to corroborate, on the one hand, the result of the orthoptic treatment, and, on the other, to develop binocular vision in cases of strabismus.

The ophthalmologist has here a vast field in which to practise his inventive power, and, let us say, it is a most fruitful field. He will succeed, in fact, much more frequently than the generally accredited opinion would lead him to hope, in re-establishing binocular vision by means of stereoscopic training.

I do not know why it seems to be believed that binocular vision is possible only for persons endowed with sensibly the same visual acuity in the two eyes, and that its re-establishment in cases of strabismus is a perfectly illusory matter.

Nothing is easier than to demonstrate that binocular vision exists, and with the greatest usefulness to the subject, in the case of persons one of whose eyes is considerably weaker than the other.

As to the re-establishment of binocular vision, we may cite the results which we have obtained by means of stereoscopic training, even in cases wherein surgical intervention only was able to overcome the ocular devia-

tion.[1] Since our original communications on this subject, cases in which binocular vision has been restored after strabotomy, in spite of great differences between the two eyes, have considerably increased in number. Nor would we ever give up these attempts at the restoration of binocular vision, since they help to complete and to consolidate the cure of strabismus.

For *emmetropes or myopes who are still young*, and who are affected by convergent strabismus, it is best to employ the same curative means as in the case of hyperopes,—that is to say, repose of the eyes, abstinence from near work, atropinization, and stereoscopic training. Rest and complete paralysis of the accommodation have, indeed, for all eyes a relaxing influence on convergence.

After having thus obtained the disappearance of the strabismus for distant vision, we make emmetropes and persons who are slightly myopic wear convex glasses strong enough to exclude all effort of accommodation during near vision. By gradually diminishing the strength of these glasses, we sometimes succeed in accustoming the patients to accommodate sufficiently without augmenting unduly their convergence. Strabismus thus cured for near vision will be all the more surely cured for distant vision.

Stereoscopic training, carried on at the same time with the use of mydriatics, according to the principles above explained, is more than ever indicated in such cases.

But if the convergent, concomitant strabismus of non-hyperopes is met with at an age when mydriatics can hardly modify the relations between convergence and accommodation, or under conditions which make illusory the effect of orthoptic treatment, it must be corrected surgically.

When convergent strabismus is due to a spasm of the convergence other than that provoked by accommodation, the weakening of the power of divergence is almost always a symptom of an affection of the nervous system. Hence it is this latter which must be combated first of all by means of a rational general treatment. In addition, this form of strabismus may be treated directly and according to the same principles which we have explained when speaking of ordinary convergent strabismus. Even atropine can sometimes render service, still more orthoptic exercise, alone at first, and then aided by operation.

## SURGICAL TREATMENT OF CONVERGENT STRABISMUS.

If, after a certain time, ordinary treatment shows itself to be inefficacious, one has a right to proceed to the *operation* for strabismus.

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[1] Landolt, International Congress of Medical Sciences, Washington, 1887; American Journal of Ophthalmology, p. 264; Arch. d'Opht., vii. p. 409; British Medical Journal, ii. p. 644, 1887; Arch. d'Opht., viii. p. 34; France Médicale, 49, 1888; Internat. Ophth. Congress, Heidelberg, 1888; Vialet, De la Cure du Strabisme dans ses Rapports avec l'Acuité visuelle de l'œil dévié; Arch. d'Opht., 289, 1890; British Medical Association meeting in Carlisle, 1896.

This "certain time" during which ordinary treatment ought to continue will necessarily vary according to a great many circumstances. One ought not to think of surgical intervention as long as an attentive examination shows a diminution in the angle of strabismus. Even if the latter remains stationary, but without exceeding a few degrees, one could still defer the operation, if the youth of the patient gave a right to hope for an ulterior development of his constitution, and if the good condition of the sight of both his eyes entitled us to count upon the powerful aid which comes from binocular vision.

It is quite another thing when the strabismus dates from long ago, when mydriatics do not modify it, and when it is concerned with a very amblyopic eye. In such cases one can proceed to operate without hesitation.

What shall this operation be? The great majority of oculists will still answer this question as it would have been answered a century ago: "It is best to do a *tenotomy*, or two, or three, or, if necessary, a reinforced tenotomy of one or both of the internal recti." [1]

Our opinion differs considerably from this routine. Since muscular setting back has been practised for the correction of convergence, experience has proved that, although the immediate effect of this operation is frequently insufficient, its final result is very often excessive. Even for the correction of convergent strabismus of twenty degrees a considerable setting back is required, one which is obtained oftenest only by means of sutures pulling the eye forcibly in the direction opposite to the tenotomized muscle. Or it is necessary in such a case to set back the internal recti on both eyes. Now, convergent strabismus of this degree is certainly not an extraordinary one, since it may amount to fifty degrees.

If the correction of the strabismus is not complete, the parents of the young patient are not satisfied, and say that the child "still squints." This objection, by itself, constitutes only a moderate inconvenience; but the young patient who to-day squints a little *inward* will squint *outward* in a very troublesome way next year, or soon afterwards. And this divergent strabismus which the surgeon has occasioned by weakening of the adductor muscles will increase in proportion as the age of the patient advances. Only slightly disfigured before the operation, he will be hideous later, especially since the divergence of the eye brings still more into notice the part of the globe which has been deprived of its muscle, and the hollow due to the retraction of the caruncle. A graver consequence of this divergent strabismus is sometimes the crossed diplopia which torments the patient, provided a still worse thing does not happen,—namely, that his binocular vision becomes and remains entirely abolished.

Even when the tenotomy of the adductors is not followed by divergent strabismus, there almost always remains a weakness of that important function, convergence.

All these facts are readily explained. We know, in the first place, that

convergent strabismus is almost always due to a spasm of the adductors, which spasm tends to diminish as age advances. Now, if by setting them back one weakens these muscles to the extent that, in spite of their contraction, the eyes have only just a parallel direction when looking at a distance, this parallelism will be changed into *divergence* in proportion as the spasm diminishes. Divergence being once established, the individual will instinctively hasten to renounce binocular vision. Only on this condition can he escape diplopia. His amplitude of convergence has, indeed, undergone a considerable reduction by the tenotomy of the muscles that bring about convergence; so that, even with the most fatiguing effort, the patient can keep up his convergence only for a limited time.

All these dangers are avoided if, instead of weakening the adductor muscles by setting them back, we increase the strength of the abductors by advancing them.[1] This method is especially indicated since, as we have demonstrated, the external recti muscles are almost always weakened in convergent strabismus.

The favor enjoyed by tenotomy in the estimation of so great a number of our colleagues is due, in the first place, to the facility of its execution, and then to that theory, as erroneous as it is inveterate, according to which the antagonist gains in power that which the tenotomized muscle loses.

Krenchel[2] has demonstrated theoretically the falsity of this theory; practice demonstrates it still more clearly; one has only to examine carefully the excursions of the eyes before and after strabotomies.

We may graphically represent the effect of tenotomy and that of muscular advancement in the following way:[3]

Let Fig. 42 be a left eye[4] affected with a convergent strabismus of thirty degrees. Let $M$ be the centre of rotation; $FS$, the false direction of the line of sight; $MN$, the normal direction; $E$, the external rectus muscle, with its insertion at $e$; $I$, the internal rectus, with its insertion at $i$.

If we would desire to give to this eye its normal direction by means only of the setting back of the internal rectus, we should be obliged to disengage this muscle considerably from its attachments; indeed, even to cause the globe to turn *by force* to the temporal side. On account of this rotation, the internal rectus would find itself strongly drawn back from the edge of the cornea. But it will be still more strongly drawn back; for, detached from the globe, this muscle will not fail to retract, according

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[1] Landolt, report of his clinic, 1878; Refraction and Accommodation, translated by Culver, 1886; International Congress of Medical Sciences, Washington, 1887; International Ophthalmological Congress, Heidelberg, 1888; Edinburgh, 1894; Arch. d'Opht., 1894; No. 8, 1895; British Medical Association, Carlisle, 1896.

[2] Krenchel, Arch. f. Ophth., xix. ii. p. 275, 1873.

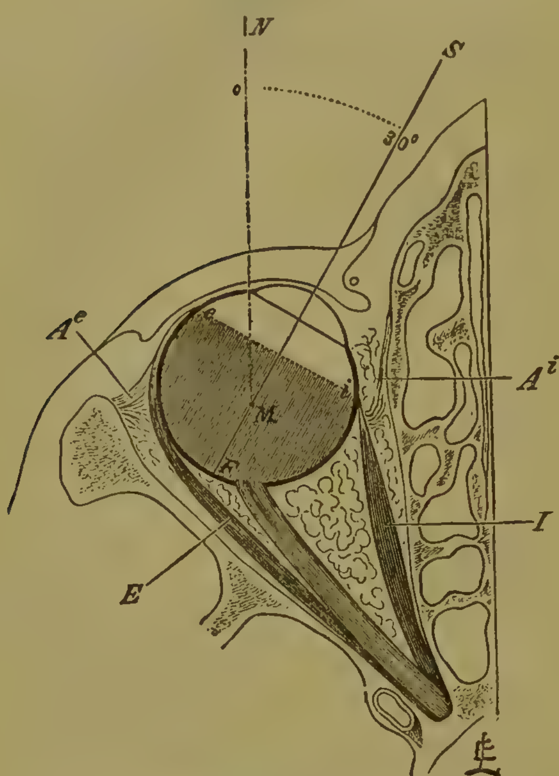
[3] See our report, already cited, to the International Congress of Ophthalmology, Heidelberg, 1888.

[4] This diagram has been made as correctly as possible. The data have been obtained from personal investigations on the cadaver, and from the anatomical drawings of Arlt and Merkel.

to its contractility and to the degree to which it has been disengaged from its attachments.

This latter factor—the retraction of the tenotomized but none the less innervated muscle—seems to have been overlooked by many operators, who took into consideration only the rotation given to the eye by the external rectus deprived of its antagonist.

Fig. 42.




Left eye. Convergent strabismus of 30°.

And this is not all; they have fallen into another grave error. In the belief that the centre of rotation of the eye is a fixed point, they have imagined that an eye deprived of the influence of its internal rectus can but rotate towards the temple round this centre. But the centre of rotation is not a fixed point in space. It results from the combination of all the forces which influence the position, the direction, and the rotation of the eyeball.[1] Among these forces are some which, as the four recti muscles, draw the eye into the back of the orbit; others, on the contrary, which tend to advance it, such as the oblique muscles and the orbital diaphragm. When one of these forces is removed, the others come more into prominence and the centre of rotation is changed. Paralysis of one of the recti muscles is sufficient to cause a certain amount of exophthalmos. During enucleation the eye falls forward gradually as the recti muscles are cut. The protrusion of an eye in consequence of a perfect tenotomy does not escape the notice of an ordinary observer, for it produces marked disfiguration. Now, this protrusion is a third reason why the tenotomized muscle is carried back behind its normal insertion, which must lessen its power over the eyeball and limit the excursion in that direction.

Does the rotation in the opposite direction in any case increase by the setting back? This is far from certain; it may even decrease.

To begin with, if we find the temporal excursion of the eye limited in a case of convergent strabismus of long standing, this limitation is due not so much to the contraction of the internal rectus as to the weakness of the external rectus consequent on want of exercise. It does not exist, indeed, at the outset of the strabismus.

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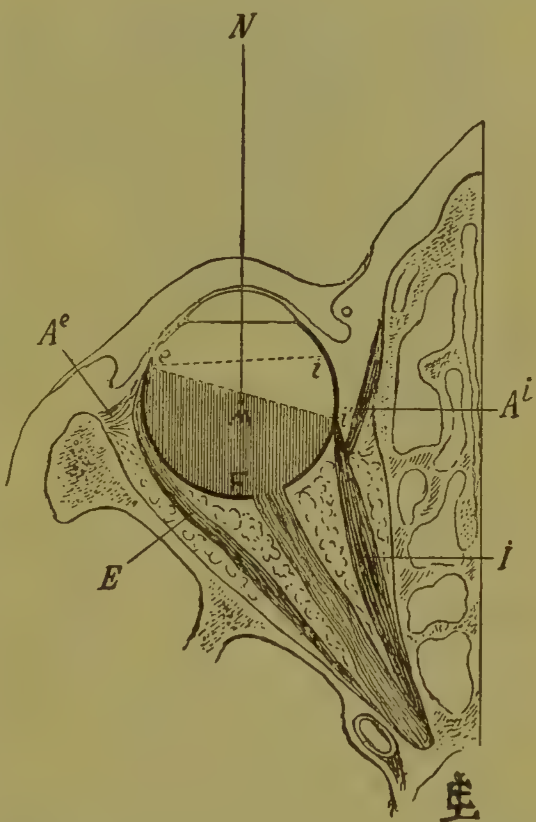
[1] Landolt, Report of the International Ophthalmological Congress, Heidelberg, 1888; British Medical Association meeting, Carlisle, 1896; Arch. of Ophth., 1897.

The injury done to the internal rectus by its setting back does not directly benefit the external rectus. On the contrary, the falling forward of the ocular globe that follows the setting back of a rectus muscle has an equally unfavorable effect upon the action of the external rectus. The advantage for the excursion on the side opposite a tenotomy is, therefore, generally not very marked. It may, indeed, be *nil*, or even a negative quantity.

Suppose that, under the influence of the three factors, the contraction of its antagonist, its own more powerful contraction, and the propulsion of the eyeball, the insertion of the tenotomized muscle is carried from $i$ to $i'$ (Fig. 43), the line $ei$ represents the limit of the muscular funnel in the horizontal meridian *before* the tenotomy, $ei'$ the limit *after* the tenotomy, and $iei'$ the amount of the protrusion of the eye, freed from the lateral muscles.

FIG. 43.




Left eye after setting back of the internal rectus.

We have seen that the action of the tenotomized muscle is considerably diminished in consequence of the withdrawal of its insertion. It is still more so by virtue of the very character of this new insertion. Indeed, the union of the tenotomized muscle with the eyeball is in most cases far from being as intimate and firm as its original attachment. Its insertion is formed by a lax cicatricial tissue, which diminishes the influence of the muscle on the ocular globe.

Lastly, the excursions of the eye are limited, even in the normal state, by fibrous expansions passing from the tendon of each muscle towards the orbital margin, $Ae$ $Ai$. (Figs. 42–44.) As they become more stretched with the retraction of the muscle, they become veritable *check-tendons*, or *check-ligaments*. (Lockwood.) This ligament, this check-tendon, follows the muscle that has been set back (Fig. 43, $Ai$), and, even though the muscle might contract still with a certain energy, the ligament would neutralize its effort all the more because the setting back of the muscle has stretched it.[1]

We thus see that numerous circumstances concur to enfeeble the retroposed muscle and to diminish the action which it exercises upon the ocular

[1] Sappay, Traité d'anatomie descriptive; Merkel, Handb. der ges. Augenheilk., i. p. 57, 1874, and Handb. der topogr. Anat., i. pp. 279 and 301; A. Graefe, Motilitaetsstoerungen, p. 152, 1880; Motais, Anatomie de l'appareil moteur de l'œil, p. 145, 1887.

globe, while its antagonist gains, so to speak, nothing positive in consequence of the tenotomy.

Hence it is not surprising to find the motility of the eye considerably limited in the meridian of the tenotomized muscle. If, for instance, the excursions have before the operation amounted to sixty-five degrees in the horizontal meridian,—that is to say, fifteen degrees to the outer side and fifty degrees to the inner side,—a corrective tenotomy may reduce the nasal excursion to twenty degrees and cause only five degrees to be gained on the temporal side, so that the total excursion will no longer amount to more than forty degrees. Hence the eye will have lost in this way twenty-five degrees of its horizontal rotation.

The field of fixation often shows no increase on the side opposite to the tenotomy, while a diminution on the same side always takes place.

What we have said concerning an extensive tenotomy is still more applicable to tenotomy reinforced by a conjunctival suture, and especially to the thread-operation of von Graefe. Such operations consist, as is known, in forcibly causing the eye to turn to the opposite side from the tenotomy, by means of the corrugation of the conjunctiva or the subconjunctival tissue, or by a thread one end of which is attached to the ocular globe, while the other is fixed at some point outside of the eye,—on the temple in our case.

For almost twenty years now we have argued against this procedure,[1] demonstrating that it may seriously injure the ocular movements in consequence of the falling forward of the globe in its muscular funnel, and of the unfavorable insertion which it gives to the muscle set back.

It is for these reasons that we recommend and practise *muscular advancement* in preference to tenotomy. Instead of enfeebling a strong muscle, we reinforce a weak one.

In the advancement of the muscle everything combines, indeed, not only to give to the eye its normal direction in the primary position, but to augment the field of its excursion.

The *operation for advancement* consists in the detachment of the muscle from its normal insertion and its reattachment by means of sutures as near as possible to the cornea. More than one method may be used for this operation. We have described the one which we actually prefer in the *Archives of Ophthalmology* (1896). The essential points are to have a raw surface for the attachment of the muscle, to make the sutures include not only the conjunctiva, but as much as possible the episclera, and to keep the patient in bed, both eyes bandaged, until firm union has taken place.

The beneficent action of muscular advancement consists, in the first place, in the augmentation of the extent of the muscle over the eyeball.

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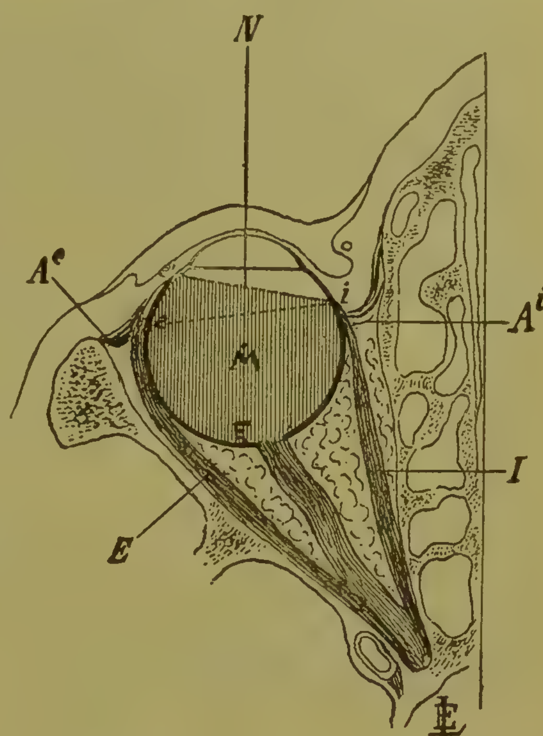
1 Landolt, Report of his clinic, 1878; International Medical Congress, Washington, 1887, p. 718 *et seq.*; British Medical Association, Dublin, 1887; International Congress of Ophthalmology, Heidelberg, 1888; International Congress of Ophthalmology, Edinburgh, 1894; British Medical Association, Carlisle, 1896.

The muscle thus has a more powerful action on the ocular globe, its insertion being brought nearer to the corneal border. It is carried, for instance, from *e* to *e'*. (Fig. 44.)

By tenotomy the eye is more or less expelled from the grasp of its muscles; the advancement, on the contrary, introduces it more deeply into its muscular funnel.

FIG. 44.




Left eye after advancement of the external rectus.

The advanced muscle is almost always somewhat shortened. We even have the habit of cutting off its tendinous extremity whenever the strabismus exceeds a certain degree; thus we still further increase the traction which the muscle exerts upon the globe of the eye.

Finally, what we have just explained concerning the influence of the check-ligaments on the tenotomized muscle is applicable, *mutatis mutandis*, to the advanced muscle. If, in the former case, the limiting action of the ligament stretched by the retracted muscle is felt very soon, in advancement the ligament advanced with the muscle permits the latter to contract to a much greater extent.

Let us consider Fig. 44. By advancing the external rectus, *E*, and thus carrying its insertion from *e* to *e'*, we have in the first place augmented by *ee'* the extent of the involution of this muscle, and consequently its rotary action on the ocular globe. We can further say that we have sunk the latter by the amount of *eic'* in its muscular funnel, which must necessarily bring about an increase of excursion in the meridian of the operation.

Again, the ligament *Ae*, whose anterior extremity has followed the tendon of the muscle in its advancement, thus permits the latter to contract more freely than in the normal state.

For all these reasons, therefore, the excursion of the eye is always increased on the side of the advanced muscle. And a peculiarly fortunate effect is that one never has to regret a diminution of the motility to the opposite side.

This fact in no way confuses us. It can only puzzle those who compare the muscles of the eye to ribbons which act in virtue of their elasticity and not of their innervation. Thanks to this strange conception and to the belief in the immobility of the centre of rotation of the eyeball, some specialists suppose that the effect of the setting back and that of the advancement of muscles are identical. According to them, in setting back

the internal rectus, what is lost in excursion on the nasal side should be gained on the temporal side. We have already shown how erroneous this idea is, and how disadvantageous the setting back is for the movements of the ball.

The converse is true of advancement. According to the theory still too popular, the temporal excursion of the eyeball should gain by the advancement of the rectus externus, but there should be an equivalent loss on the nasal side, because, as is said, the internal rectus has now a stronger antagonist. But one forgets that the external rectus opposes the internal only when it contracts, and it contracts only in answer to a nervous stimulus. Otherwise it is more or less limp and does not prevent the eyeball from turning in the opposite direction. So, in spite of the advancement of the external rectus of the left eye, for example, that eye will be able to turn towards the right as well as before the operation. Or why should the left external rectus contract when the eye turns to the right side?

The same holds good for convergence. This function is not injured by the advancement of the external recti, because the nervous stimulus for convergence is directed essentially towards the internal recti and not towards the abducting muscles.

In one word, in advancing a muscle, we gain on that side because we have won more favorable conditions for its action, and we lose nothing on the other side because we have left its antagonist intact. As a matter of fact, experience bears us out, and if the excursions of the eyes were measured carefully before and after the various operations for strabismus, there would be less discussion among the surgeons and less deception among the patients.

We practise muscular advancement alone in low and medium degrees of strabismus. If the deviation is one of only a few degrees, it may suffice to advance the external rectus of the eye which usually converges; but it is generally better to perform the same operation on both eyes at the outset. This way of doing is more logical than that which seeks to obtain the whole correction by an operation on a single eye, because, as we have pointed out, concomitant strabismus is a binocular affection.

Experience justifies our contention, moreover, by showing that this double advancement, aided by the treatment which has been explained in the preceding paragraph, gives perfect results without any of the disadvantages of tenotomy.

Above all, we have never found occasion to regret the over-correction which occurs only too frequently after tenotomy. It is for that reason that we always advance the muscle as near as possible to the edge of the cornea. Nothing is easier, as we shall see, than to diminish the effect of this operation, especially in convergent strabismus.

As to the technique of muscular advancement, it is the subject of another article of this System. It is not our jurisdiction to cite the multiple

and varied methods of it.[1] For the same reason we only mention the operations which, like *capsular advancement* (von Wecker) and the *folding of the muscle* (Knapp), act as weak advancements.

What we have said in favor of muscular advancement must not be understood as implying condemnation of tenotomy. Although we never practise setting back alone in convergent strabismus, but give preference to advancement, even in strabismus of low degree, in certain cases of insufficiency of convergence or difference in height between the two eyes, tenotomy, cautiously performed and combined with orthoptic training, may give satisfactory results.

Sometimes we also have recourse to tenotomy in order to increase the effect of the advancement. But this is necessary only in the highest degrees of strabismus, and especially in paralytic strabismus.

Even in such cases, however, the muscle must not be set back very far. The conjunctival incision should be short, parallel with the muscle, in order to avoid retraction of the caruncle, and the muscle should be disengaged from its adhesions only to a very moderate extent. In a word, the advancement ought always to predominate over the setting back of the muscles. And since it is not possible to foresee the ultimate effect of any strabotomy, we advise not to perform both operations at the same sitting, but to await the effect of the double advancement before practising a tenotomy.

After any operation for strabismus, binocular bandaging is the rule. We have already set forth in our communication to the Congress at Washington[2] that "Just as strabismus is a binocular affection, the operation for strabismus is a binocular operation," because, although it is performed on only one eye, it nevertheless always affects both.

Binocular bandaging is peculiarly important after the operation for *convergent* strabismus. Darkness, inactivity, the absence of any object that might provoke an effort of convergence, become in this case, with atropinization, powerful orthoptic therapeutic agents.

If the effect of the operation should seem excessive, atropine may be suppressed; but one is not to be frightened by even a marked divergence after advancement. It is formidable only when the internal rectus has been tenotomized.

When divergence follows tenotomy, the muscle which has been set back should at once be sought for and advanced by a firm suture. This manœuvre is easy, especially with our forceps, the jaws of which are oblique. (Fig. 45.) The instrument is therefore well adapted to catch the muscle for its tenotomy, and is no less useful for finding the set-back muscle. In the latter case one has only to reverse the forceps, so that its jaws are directed upward towards the conjunctiva.

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[1] Vide our method in the Archives of Ophthalmology, xxvi., No. 1, 1897.

[2] Loco citato, p. 718.

If the over-correction is due to advancement, it nearly always disappears entirely of itself and without the least prejudice to the positive part of the amplitude of convergence. The negative part—that is to say, divergence—is always augmented, and thus very favorably counterbalances the spasm of the adductor muscles.

Fig. 45.


Landolt's forceps.

As to the excursions of the eyes in the associated movements, we have, as said before, always found a considerable increase on the temporal side of the field of fixation without any loss on the opposite side.

Hence in case of apparent over-correction after advancement one need not hurry either to do away with the binocular dressing or to remove the sutures. For the most part we let the threads remain six days and the dressing about a week, unless peculiar circumstances oblige us to leave one or both eyes unbandaged sooner.

The surgical dressing is immediately replaced by lenses which correct the total hyperopia. These lenses should be large and of smoked tint, in order to shield the eyes from the dazzling resulting from the mydriasis.

It is best, indeed, to continue the orthoptic treatment for a long time. The operation, by itself, by no means cures the strabismus; it is only a part of the treatment, a powerful aid lent to it. If we do not complete the cure by exercises tending to re-establish binocular vision, by the training of convergence and divergence, we shall never get a satisfactory result, or if we accidentally obtain it, we may soon lose it again.[1]

It would take too long to enumerate here all the difficulties which may be met with in the surgical treatment of strabismus. We cannot, however, remain silent concerning a somewhat frequent peculiarity which is due to the disturbance of "orientation" in the deviating eye. It is a new proof of what we have always held, that the most perfect surgical correction is not always sufficient, even if the vision is good on both sides, to re-establish binocular vision. The eyes have not only forgotten—if they have ever learned—to fuse the impressions of their foveæ, but the visual centre of the deviating eye may so accustom itself to the vicious direction of this eye that its retinal images are *falsely projected.* It happens, indeed, as we have said above, that the part of the retina of the deviating eye, which receives the image of the object that is fixed by the normal eye, usurps to a certain degree the functions of the macula. It succeeds not only in fusing its impression with that of the other eye, but it becomes the *centre of orientation* for the deviating eye.

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[1] Landolt, Le résultat idéal de l'opération du Strabisme, Société de Médecine pratique, 27 Juin, 1889.

Thus it is that one sometimes notices, after a perfectly successful operation for *convergent* strabismus, that the patient complains of a strong crossed diplopia, as if he were affected with a high degree of divergent strabismus. If his former convergent strabismus amounted to twenty degrees, a part of the retina situated twenty degrees to the internal side of the macula had become almost equivalent to this macula. By the straightening of the eye the physiological macula has taken its normal place opposite the fixation-object, and the "false macula," as it has been called, like all other points of the horizontal meridian, has been carried twenty degrees inward. Now, if the patient continues to consider this latter part as the centre of his retina, he finds himself like one who is affected with *divergent* strabismus, and if he projects the retinal images of this eye accordingly, the image received at the anatomical macula will be referred to an object situated at the nasal side, where it would have needed to be in order to form its image on the macula while the eye was still deviating inward. In a word, one observes in this case a crossed diplopia analogous to that of paralysis of the internal rectus, and of a degree equal to that of the pre-existing convergent strabismus, if the correction has been perfect, or less if it has been less.

On the other hand, it may happen that the eye cured of strabismus by operation projects at once with reference to the two centres of orientation. It sees the object whose image is produced in the physiological macula at the same time in its true position and on the nasal side,—that is to say, with monocular diplopia. Moreover, if neither of these two images is fused with that of the other eye, *triplopia* may result. Or, again, the two kinds of projections may alternate.

Monocular diplopia rarely persists. For the most part, the normal macula promptly resumes its function, especially if the operation has been correctly performed and if the stereoscopic training follows and completes its effect.

The same thing that we have just pointed out may likewise be produced in *divergent strabismus.* The diplopia which follows the operation is in this case *homonymous,* the false macula being carried towards the temple by the straightening of the eye.

Correct fusion and binocular vision being finally re-established, we cannot abandon our patient. We must guide him for some time yet in the use of his recovered eyes.

According as the tendency to convergent strabismus is more or less abolished, and according to the age of the patient, we make him wear convex lenses correcting the manifest hyperopia (when mydriatics have become superfluous) either constantly or for near work only.

We do away with them entirely if there is any tendency to divergence.

We should resume, on the contrary, all the treatment of strabismus—atropinization, correcting glasses, and repose of the eyes—if the convergence reappears.

In this way one continues master of the situation until the strabismus is completely and radically cured. The result is then so perfect that not only does the individual enjoy all the advantages of recovered binocular vision, but there remains no vestige of the operation, unless freer excursions and an amplitude of convergence which is more extensive even than in the normal condition.

### NON-PARALYTIC DIVERGENT STRABISMUS.

As a rule, *the eyes in a condition of complete repose diverge.*[1]

There is nothing surprising in this fact; we have only to consider that the orbits diverge from each other at an angle of about forty degrees.[2] (Fig. 10.) The abductor muscles encircle the ocular globes for a considerable distance, as if they had followed the eyes in their forward rotation, and have almost an extended appearance owing to the exercise of convergence, a function which is an acquisition of the highest vertebrates only. The lower one descends in the series of vertebrates the more the eyes diverge, until finally they are found to be diametrically placed on either side of the head, as in certain fishes.

However high the human race may be elevated above its ancestors, convergence still constitutes an effort. Abandoned to themselves, the eyes diverge. This is a law which admits of few exceptions.

Convergence was evolved, of course, in the interest of *binocular vision*. It is, moreover, so intimately associated with *accommodation* that under normal circumstances an effort of accommodation suffices to provoke a convergence movement.

However, if binocular vision and accommodation are lacking, the eyes place themselves almost invariably in divergence. That is why an amblyopic eye—an eye that is useless—almost always deviates towards the temple, and why myopes, who exercise their accommodation much less than emmetropes or hyperopes, furnish the large majority of divergent strabismus.

In myopia of high grade, divergent strabismus is the rule. The ellipsoidal form which such eyes assume, and which is the cause of their ametropia, of itself tends to make them take the direction of the orbits, in which they are somewhat closely lodged.

Their movements, especially that of convergence, are hindered, moreover, by the peculiar form of the myopic eyeball. They are still further limited in consequence of the elongation of their muscles; for, as Fuchs[3] has demonstrated, only that portion of the globe which is posterior to the insertion of the muscles participates in the elongation of the eye which produces myopia.

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[1] See p. 19.

[2] According to Merkel, forty-two or forty-four degrees; Handb. der topogr. Anatomie, i. p. 237.

[3] Fuchs, Archiv für Ophthalmologie, xxx. 4, 1884, 1.

According to Donders, the angle *gamma* is particularly small in myopes. It may even become *nil* or negative,—that is to say, the line of sight may pass *outside* the optic axis.[1] We have found, as have also Dobrowolsky and Botto, that the angle epsilon comprised between the papilla and the fovea centralis (with its apex at the second nodal point) is likewise smaller in myopes, and that often, in spite of the increased volume of the eyeball, the distance between these two points is less than in emmetropia. It is evident that the nearer the macula is to the axis of the globe the greater must be the effort of convergence to direct the lines of sight towards the object of fixation.

Hence it is seen that in this form of myopia several circumstances concur to render convergence difficult. This difficulty of convergence is the more troublesome, as myopes of high degree are obliged to bring the object very near in order to see distinctly. They are thus all the less apt to converge as they have more need of convergence in order to see binocularly.

Finally, by virtue of their static refraction such myopes have no need of accommodation in order to see distinctly even at a very short distance. The contraction of the ciliary muscle which causes the emmetrope to converge, even when one of his eyes is excluded, and which in hyperopia brings about exaggerated convergence, is lacking in a myope whose ametropia is of high degree. The convergence rendered difficult by the form of the eyeball is not excited by the accommodation.

It is evident that under such circumstances binocular vision is either not developed or, at best, is developed in but a rudimentary way. Indeed, there is an advantage to the individual in suppressing as completely as possible the visual impression of the eye which does not fix, in order not to be troubled by the crossed diplopia resulting from the divergence of his visual lines.

A person affected with a high degree of myopia brings the object within the region of the distinct vision of his better eye without making an effort of convergence. The other eye diverges. Even if the eyes were parallel, this parallelism would of itself represent a considerable *relative* divergence, since binocular vision would often require a convergence of ninety degrees or more. But for the most part the divergence is *absolute*. It exists for all distances and for all directions of the gaze.

That which happens inevitably in extreme myopia very often occurs also in the case of myopes of less degree.

In fact, the normal relation which exists between convergence and accommodation ought to undergo, in the case of a myope, a considerable alteration, since he sees without any effort of accommodation at a distance for which he is obliged to use a comparatively great amount of convergence, —*e.g.*, for his *punctum remotum*.

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[1] Landolt, Refraction and Accommodation, p. 116.

Thus, a myope of 4 D. sees clearly without accommodating at one-quarter of a metre (or twenty-five centimetres). But in order to see simply he must make a convergence effort of 4*ma*. For all distances situated nearer than his *punctum remotum* the accommodation is less by four units than is the convergence.

If the myope possesses an amplitude of positive relative convergence[1] of 4*ma*, binocular and single vision will be possible. If he does not have such latitude in his converging power,—if, in a general way, the degree of the myopia surpasses that of the amplitude of positive relative convergence, —there exists an *insufficiency of convergence*, unless the accommodation should enter into play in an exaggerated way, the effect of which would be to render vision indistinct.

There are, of course, myopes the degree of whose ametropia is slight enough, or whose amplitude of positive relative convergence is sufficiently developed, for binocular clear vision to be possible at any distance between the *punctum remotum* and the *punctum proximum* without the use of correcting glasses. But the independence of convergence relatively to accommodation is limited, and does not admit of a very high degree of ametropia. Beyond 5 D. discord between the two functions is easily established.

It is true that this lack of harmony does not by itself constitute divergent strabismus. There exists only a *relative* divergence of the lines of sight for all points situated nearer than the *punctum remotum*, or rather a tendency to relative divergence, which is manifest only when one eye is occluded or when binocular vision is made impossible by the use of a vertical prism or some other means.

In virtue of this incongruity between the two functions, however, convergence usually develops poorly with such persons; for, unless endowed with a peculiarly great amount of converging power, the individual may find it advantageous to disregard the image received by one of his eyes for the sake of escaping diplopia; in a word, binocular vision may not acquire a preponderant influence upon the relative direction of the eyes.

If there be added to this a defective development or an enfeebling of the motor muscles, in consequence of a debilitating illness, the individual will only too promptly abandon the convergence which is troublesome to him. Let one of his eyes, for any reason, be inferior to the other, and he will no longer make an effort of convergence which neither binocular vision nor accommodation requires of him.

Thus we see that if the typical convergent strabismus—that of hyperopes—is an *active*, a *spastic* strabismus, divergent strabismus is an essentially *passive* strabismus, due to a relaxation or to a lack of development of convergence.

Although myopia, for the reasons just stated, furnishes the largest proportion of cases of divergent squint, yet it is found also in other states

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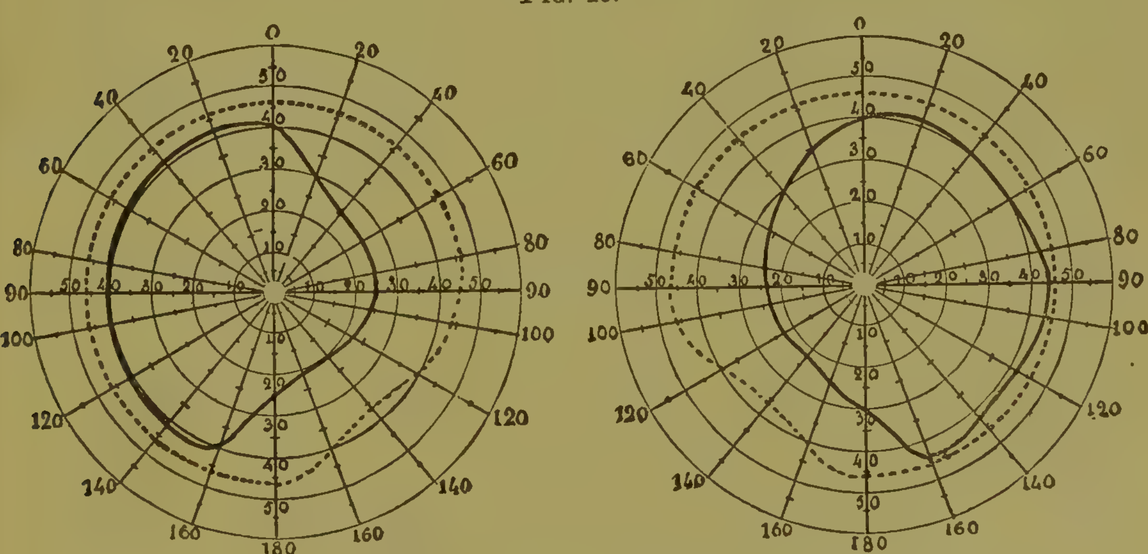
[1] Landolt, Refraction and Accommodation, pp. 196–218.

of refraction. We have already said that any cause which abolishes binocular vision predisposes to strabismus. Now, the most natural deviation of the eyes is evidently divergence; it almost always corresponds to their position of repose, and consequently is accomplished without effort. It is for this reason that divergent strabismus is the rule in cases of monocular amblyopia or amaurosis.

This fact is usually explained by saying that the amblyopic eye, not being able to collaborate usefully with its congener, becomes disinterested, so to speak, in vision, follows its own course, and deviates outward. This explanation seems to correspond with what happens daily, when we see divergent strabismus established, often in the course of a few months, in an eye which has suddenly become blind. However, this is not the proper way in which to consider this phenomenon. To be correct, we should say, binocular vision being abolished, the eyes no longer make the effort of convergence, which effort is useless and troublesome to them; they abandon themselves to a relative divergence, which gradually becomes absolute. Only, as one of the eyes is always directed towards the object to be fixed, the deviation manifests itself exclusively in the amblyopic eye. But

Fig. 46.




Fields of fixation of two strongly myopic eyes affected with divergent strabismus.

divergent strabismus is a *binocular* strabismus just as is convergent strabismus.[1]

In the same way as the examination of the field of fixation denotes in convergent strabismus of long standing a limitation of the temporal excursions in *both* eyes, so the excursions towards the nasal side are always found limited in *both* eyes, although only one, and always the same one, is the victim of *divergent strabismus.*

This defect in motility is in both cases, in the majority of instances, not the cause but the consequence of strabismus. In divergent strabismus it is due to a lack of use of the adductor muscles, for which convergence

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[1] Landolt, Archives d'Ophtalmologie, 1897, p. 85.

constitutes an exercise much more powerful than do the associated lateral movements.

Whenever we have had to speak of the relations *between accommodation and convergence*, we have taken care to remark that these relations have nothing absolute about them, and that consequently one cannot admit a single centre of innervation as ruling them simultaneously. This conclusion is proved by the classical experiments of Donders, as well as by our own, on the relative amplitudes of accommodation and of convergence; experiments which have demonstrated to what degree the two functions may be exercised independently of each other.

This independence, moreover, is shown by the fact that the amplitude of accommodation gradually diminishes as age advances until it becomes *nil*, while convergence remains almost invariable during the whole life. Hence the relations between the two functions must be constantly modified; otherwise binocular and distinct vision would not be possible even for emmetropic eyes. The individual must learn how to associate the same degree of convergence with a greater and greater degree of accommodation.

That, however, which is produced in one direction, for the sake of binocular vision, can quite as well be produced in the opposite direction, for the sake of the individual's comfort, when binocular vision is lacking. As long as the emmetrope fuses well the visual impressions of his two eyes, accommodation causes him to converge so correctly that he directs even the eye that has been excluded from vision towards the object fixed by the other.

But if he loses the sight of one of his eyes, he learns equally well to abandon the effort of convergence which is henceforth useless, although accommodating correctly. And if he notices that this insufficient convergence no longer brings about, as it formerly did, the visual trouble resulting from the diplopia, nothing is more natural than that the convergence should be almost entirely abolished.

What is produced in the case of the emmetrope may equally well happen to the hyperope, although those among them who renounce binocular vision do so more often by augmenting convergence in order to facilitate accommodation. However, we have said before that this stratagem could not succeed with all hyperopes. Indeed, not all of them are condemned to convergent strabismus. Some of them, deprived of binocular vision, squint outward and furnish a certain contingent (five per cent., according to Schweigger) of divergent strabismus.

Just as in the case of convergent strabismus, *local circumstances* have been invoked as capable of favoring the production of divergent strabismus. These have been supposed to be found in a *vicious insertion*, a *defective development*, an *insufficient power of the adductor muscles*, or, again, in the *divergence* or in the *excessive separation of the orbits*. *Smallness of the angle gamma* has likewise been brought forward to explain the genesis of divergent strabismus among non-myopes. According to Stilling and others,

certain eyes ought to be predisposed to divergent strabismus by their *position of equilibrium*, which, in these cases, is divergence. Another theory holds that it is a *primary lesion of the centres* controlling the symmetrical movements of the eyes which is the cause of this form of strabismus.

We have already mentioned these various attempts to explain the anomalies of the relative direction of the eyes when discussing the etiology of convergent strabismus. We shall not further review the matter here, except to repeat that, although strabismus usually permits of a more satisfactory explanation, it is well to take all these circumstances into consideration.

Divergent strabismus is often at first *relative*,—that is to say, it manifests itself only under certain circumstances. While looking at a great distance, for instance, the eyes may be normally directed towards the object of fixation, especially when they are furnished with correcting glasses. Even if the object be brought nearer, they still follow it perfectly up to a certain point; but the attentive observer notices then that convergence seems no longer to increase proportionately with the approach of the object.

Only one eye fixes it; the visual line of the other eye passes farther and farther from the object. Then comes a moment when this latter eye seems to hesitate as to whether it ought to converge or not; it still makes a few spasmodic efforts, then abandons the contest and deviates outward. This phenomenon is, on the whole, analogous to that which we can prove at any time on ourselves when the fixation-object approaches the *punctum proximum* of convergence. The only difference is that in pathological cases these phenomena appear sooner, because the *punctum proximum* of convergence is farther away, the convergence feebler.

It is noticeable also in such cases that, even when both eyes are directed towards the object of fixation, they diverge as soon as binocular vision is made impossible, whether by covering either of the eyes or by the use of a vertical prism. This beginning of divergent strabismus is sometimes accompanied by asthenopia, just as is insufficiency of convergence. It is, in fact, the same thing. The patients complain of headache, of vertigo, of all sorts of visual disturbances which torment them during near work. These phenomena are due to the exaggerated effort necessary for convergence, and also to the diplopia which manifests itself whenever convergence can no longer be maintained. It is true that the patients rarely complain of *double* vision; they complain of seeing *confusedly;* letters seem to run into each other, lines move, become confounded with each other, etc. But when we analyze their sensations we recognize that they are very often due to the reduplication of the letters looked at.

This initial stage of divergent strabismus may endure for a variable period,—months or years. When there is myopia of somewhat high degree, the relative strabismus only too promptly becomes absolute, especially if one of the eyes be weaker than the other. It is established especially early in cases of amblyopia of one eye, whether or not the other be myopic.

However, one must guard against considering every divergence that is met with in an amblyopic eye as concomitant strabismus. Just as convergent strabismus may be due to paresis of one or both of the abductors, so divergent strabismus may have the paresis of an internus for its cause. The latter is evidently much rarer than the former, inasmuch as the internal rectus, belonging to a muscular group which is innervated by the third pair, is only very exceptionally alone the victim of paresis. Such cases do present themselves, however, and the rarer they are the more important it is to give them attention, so as to know how to recognize them.

The differential diagnosis between concomitant and paralytic divergent strabismus might seem to be very simple. It is so in typical cases, where a well-defined diplopia increasing in one direction and diminishing in the other leaves no doubt as to the paralysis of an ocular muscle, while the absence of diplopia, the equality of the conjugate movements of the two eyes, the gradual development of the deviation, etc , characterize concomitant strabismus. But there exist cases in which the absence of binocular vision, because of the amblyopia of one eye, excludes diplopia,—that is to say, the most striking symptom of paralytic strabismus. In such a case we have recourse to measurement of the excursions of the eyes. If there be paralysis, limitation of the field of fixation will soon teach us not only the nature of the strabismus, but also which muscle is implicated.

But when the strabismus is not very pronounced, the limitation of the field of fixation is often not sufficiently characteristic to establish the differential diagnosis.

I remember a case of divergent strabismus of this kind which was all the more puzzling because it occurred in a girl sixteen years old,—that is to say, at an age when muscular pareses are rather rare. One of her eyes was myopic and amblyopic, the other emmetropic and endowed with good visual acuity. The divergence of the defective eye seemed quite natural, according to the etiology of this kind of strabismus which we have just given.

One thing, however, which was striking about this case was a power of convergence extraordinarily well developed relatively to the degree of strabismus. When the patient's attention was attracted energetically to a near object, she succeeded in converging in a truly astonishing way. Since binocular vision was lacking, it was impossible to ascertain whether or not the convergence was correct.

I then covered the good eye and made the patient fix a candle-flame with only the squinting eye. What was at first very strange was that the patient, instead of directing the eye promptly towards the object, left the eye in the divergent position and declared that she saw nothing. However, on stimulating her visual energy, I succeeded in making her direct the diseased eye towards the object; but, on asking her at what point she saw the object, she showed us by extending her hand, not in front of her, but in the direction opposite to her strabismus. Hence there was *false projection.* This false projection is, as I have above explained, due to a disproportion between the innervation brought into action and the effect of the muscular contraction. It establishes a paresis of the muscle, which, to effect a given rotation of the eye, requires a larger amount of innervation than in the normal state.

This defect in orientation, this erroneous localization of objects fixed by the imperfect eye, was again manifest when I induced the patient to try to touch rapidly a pencil-point placed before her.

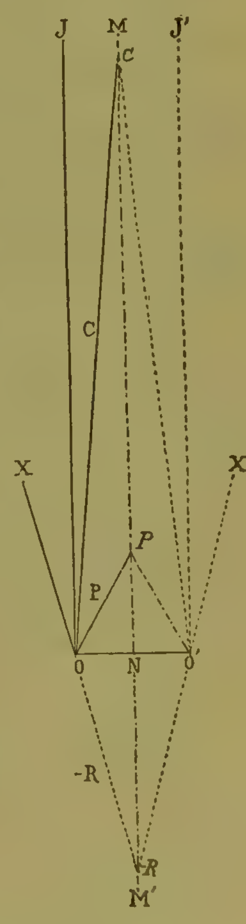
Thus, when from the form of the diplopia and from the examination of the movements of the eyes we learn little or nothing as to the nature of the strabismus, then the projection—the muscular sense—may furnish us with decisive indications with which to establish the differential diagnosis between concomitant and paralytic strabismus.

When the deviating eye is amaurotic, or when it has acquired the habit of fixing with an eccentric part of its retina, this symptom is evidently lacking; but the strabismus is generally very marked in such a case, and when one examines the field of fixation, which is possible, even for an amblyopic eye, by means of the corneal reflex, one almost always finds a limitation in both eyes in concomitant strabismus, while the excursion of the deviating eye is much more limited in paralysis.

### INSUFFICIENCY OF CONVERGENCE.

In the beginning of this chapter we considered the movements that the two eyes are accustomed to execute together for the requirement of binocular vision. Whatever may be the origin of the nerves controlling these movements, whatever may be the muscles which participate in them, clinical study has shown that the associated movements may be altered as such. Let us recall merely the paralyses of the associated lateral movements and those of the raising and lowering of the eyes. The same thing is produced much more frequently still for the adduction of the eyes,—*e.g.*, for *convergence.*

FIG. 47.




Although von Graefe, A. Graefe, Horner, and others had already known and pointed out a form of asthenopia due to the impossibility of converging sufficiently, it was Krenchel[1] who in 1873 clearly established that *convergence* must be considered a function by itself, a function which may be altered even without lesion of the muscles which produce it, the excursions of each of the eyes being otherwise perfectly intact.

Stevens, Parinaud, Moebius, de Watteville, Gowers, G. Steward, my students, Huebscher and Borel, and myself have given many and varied observations in support of the correctness of this method of regarding it, so that *insufficiency of convergence* now appears as a well-defined morbid entity.

In order to recognize and to treat the alterations of the power of convergence we shall have to study this function more closely.

I have designated by the term *convergence*[2] the faculty of directing the two eyes towards the object of fixation, whether this object be situated at a

[1] Krenchel, Ueber die krankhaft herabgesetze Fusionsbreite als Ursache des Schielens, Arch. f. Ophth., xix. 1, S. 142, 1873.

[2] Landolt, Insufficienz des Convergenzvermögens, Heidelberg meeting, 1885, and Refraction and Accommodation, translated by Culver, Edinburgh, 1886.

finite distance, at infinity, or even beyond infinity,—that is to say, whether its fixation requires actual convergence, parallelism, or divergence.

The most natural method of measuring convergence is by means of an object of fixation placed in the median line ($MM'$, Fig. 47). In this way the *angle of convergence* is always the same for each eye. This angle evidently gives the measure for the convergence effort put forth by each eye.

In distant vision, the two eyes ($O$ and $O'$) having a parallel direction, this angle is *nil*. It increases in proportion as the object fixed is brought nearer. It may be said, then, that the angle of convergence is in *inverse* proportion to the distance between either eye and the fixed object in the median line.[1]

If the object be at $C$ at the distance $OC = \mathrm{C}$, the angle of convergence $JOC$ can be expressed by $\mathrm{c} = \frac{1}{\mathrm{C}}$.

If we measure this distance C by the aid of the *metre*, we obtain for the convergence required in binocular fixation an expression identical with that for the refraction necessary for distinct vision of the same object.

Thus, supposing an object to be situated at a distance of one metre from each eye, there must be for both eyes $\frac{1}{1m} = 1$ dioptry of positive refraction and $\frac{1}{1m} = 1$ unit of positive convergence.

This unit is called, after Nagel, to whom we are indebted for this principle of measurement, the *metre-angle*.[2]

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[1] In reality this ratio is not the *angle* of convergence, but its sine. For our purpose, however, we may conveniently substitute one for the other.

[2] This expression is analogous with *metre-lens*, which Nagel has proposed for the dioptry. The *absolute* value of the metre-angle depends upon the distance (base-line) which separates the centres of rotation of the eyeballs from each other. For instance, if this base-line is 58 millimetres in length, the metre-angle is = 1° 39′ 39″; say 1° 40′, or 100′. For a base-line of 64 millimetres the metre-angle is = 1° 50′; say 110′, and so on. (Nagel, in Graefe-Saemisch Handbuch, vi. p. 478.)

The following table gives the equivalents between degrees and metre-angles (M.A.) for the two values of the base-line.

| Base-Line. 58 mm. | | | Base-Line. 64 mm. | | | Base-Line. 58 mm. | | | Base-Line. 64 mm. | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Degrees. | | M.A. | Degrees. | | M.A. | M.A. | | Degrees. | M.A. | | Degrees. |
| 0.5 | = | 0.3 | 0.5 | = | 0.27 | 0.5 | = | 0° 50 | 0.5 | = | 0° 55 |
| 1 | = | 0.6 | 1 | = | 0.55 | 1 | = | 1° 40 | 1 | = | 1° 50 |
| 1.50 | = | 0.9 | 1.50 | = | 0.82 | 2 | = | 3° 20 | 2 | = | 3° 40 |
| 2 | = | 1.2 | 2 | = | 1.09 | 3 | = | 5° | 3 | = | 5° 30 |
| 2.50 | = | 1.5 | 2.50 | = | 1.36 | 4 | = | 6° 40 | 4 | = | 7° 20 |
| 3 | = | 1.8 | 3 | = | 1.64 | 5 | = | 8° 20 | 5 | = | 9° 10 |
| 4 | = | 2.4 | 4 | = | 2.18 | 6 | = | 10° | 6 | = | 11° |
| 5 | = | 3 | 5 | = | 2.73 | 7 | = | 11° 40 | 7 | = | 12° 50 |
| 6 | = | 3.6 | 6 | = | 3.27 | 8 | = | 13° 20 | 8 | = | 14° 40 |
| 7 | = | 4.2 | 7 | = | 3.82 | 9 | = | 15° | 9 | = | 16° 30 |
| 8 | = | 4.8 | 8 | = | 4.36 | 10 | = | 16° 40 | 10 | = | 18° 20 |
| 9 | = | 5.4 | 9 | = | 4.91 | 11 | = | 18° 20 | 11 | = | 20° 10 |
| 10 | = | 6 | 10 | = | 5.46 | 12 | = | 20° | 12 | = | 22° |

If the object is placed at one-third of a metre from each eye, $\frac{1}{1m/3} = 3$ D. and $3ma$ are required, and so on.

The *amplitude* of convergence is obviously contained between the maximum and the minimum of the convergence which an individual is capable of exerting.

The *maximum* of convergence is inversely as the distance of the nearest point, *punctum proximum of convergence,* which can be fixed binocularly. If P be the distance which separates this point from each eye, the maximum of convergence is $= \frac{1}{P}$. In measuring the distance P by means of the metre, we can replace this fraction by the value of p metre-angles.

The *minimum of convergence,* upon the same principle, is inversely proportional to the distance which separates each eye from the *farthest point* which can be fixed binocularly. If R be the distance of this *punctum remotum of convergence,* the minimum of convergence will be $\frac{1}{R} = r\,ma$.

If this latter point be situated at a *finite* distance, the minimum of convergence is *positive,* and can be determined in the same manner as the maximum, as I shall hereafter explain. But this happens only in pathological cases. Under normal conditions the lines of fixation can be directed at least parallel with each other. The minimum of convergence in such a case is equal to zero, because the *punctum remotum* is situated at infinity and $r = \frac{1}{\infty}$ 0.

Most normal eyes, however, can *diverge* more or less. The minimum of convergence is then *negative.* It is always inversely proportional to the distance of the *punctum remotum,* only, as the lines of fixation diverge, this point is situated not in front of the head, but behind it (—*R,* Fig. 47), where the lines of fixation, prolonged backward, meet.

The *amplitude of convergence* (a) is represented by the difference between the maximum and the minimum of this function :

$$a = p - r.$$

In normal cases I have found that, on the average, the minimum of convergence is about —$1ma$, the maximum $9.5ma$, and the amplitude of convergence, therefore, $10.5ma$.

When the minimum of convergence is *negative,* its amount is measured by the strongest abducting prism which can be overcome in distant vision. We have, however, to bear in mind that, although the prism be placed before one eye only, it acts, notwithstanding, upon both. Hence the value of the prism has to be divided by 2, in order to obtain its deviating action for each eye.

If the prism is numbered—as is desirable—according to the angle of the deflection of light which it produces, it suffices (for *children* whose base-line equals 58 millimetres and 1 metre-angle = 100′) to multiply this number by 3 and to divide the product by 10, in

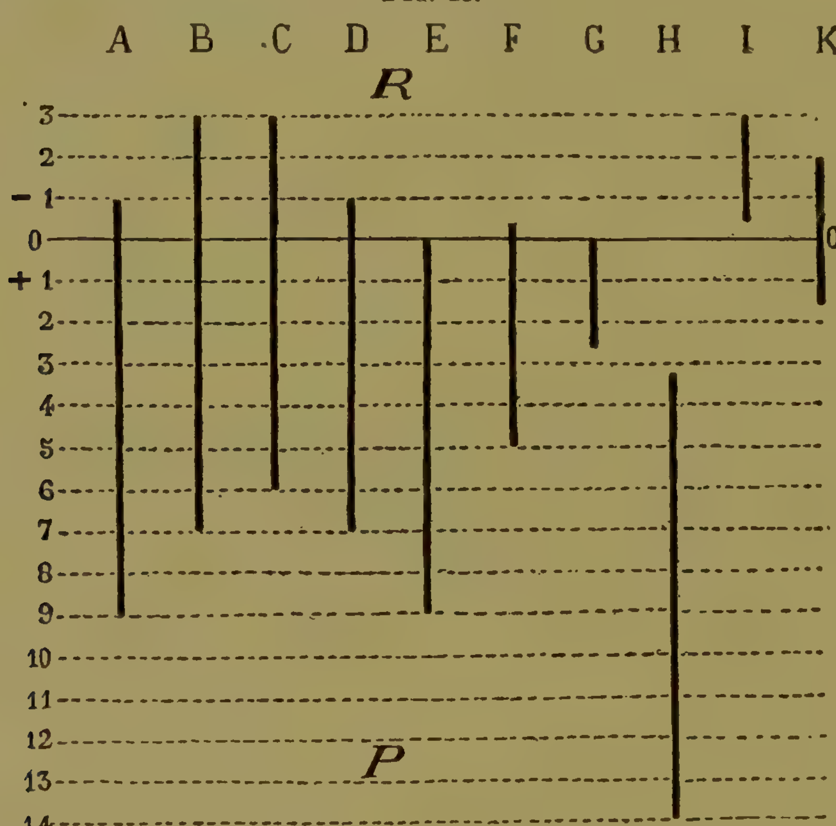
order to obtain the corresponding number of metre-angles. Thus, a prism of five degrees will correspond to $\frac{5 \times 3}{10} = 1.5ma$.

For adults, in whose case, because of the greater separation of the eyes (64 millimetres), the absolute value of the metre-angle is greater (110′), the formula becomes $\frac{x.3}{11}$, if we designate by $x$ the number of degrees to be converted into metre-angles. Five degrees correspond, in this case, to $\frac{5 \times 3}{11} = 1.3ma.$[1]

In the normal condition positive convergence ought to amount to *at least* $9ma$; in other words, the eyes ought to be able to fix easily an object $P$ (Fig. 47) situated at a distance of $\frac{1m}{9} = 11$ centimetres from them.

On the other hand, they can diverge about one metre-angle,—that is to say, to such an extent that their lines of sight meet at $-R$ (Fig. 47), one metre behind the head.

FIG. 48.




We can represent the amplitude of convergence graphically, as is shown by Fig. 48. In this diagram the range of convergence is represented

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[1] If the prism be numbered according to the method formerly used,—that is to say, according to its apex-angle,—we have to divide the number of the prism by 7 to obtain for each eye, in metre-angles, the rotation required to overcome it. The distance between the two eyes, in this case, is admitted to be between 58 and 64 millimetres. A prism of No. 14 held horizontally before *one* eye requires from *each* eye a rotation of $\frac{14}{7} = 2ma$ *adduction* if the apex of the prism is turned inward, *abduction* if the prism-apex is turned outward. A person who, during distant vision, can overcome an abducting prism of old No. 5 has a divergence power $-r = \frac{5}{7} = .71ma$.

by vertical lines. The full horizontal line indicates zero *ma,*—that is to say, it corresponds to parallelism of the lines of sight.

The part *above* the horizontal represents *negative convergence* (or *divergence*), the part *below* the horizontal represents *positive convergence.*

The figures and dotted lines indicate metre-angles. Hence the line A represents the normal condition of the amplitude of convergence, the positive part p = 9 ; r = —1*ma.*

It might appear that nature, which in the distribution of our functions is generally so parsimonious, had been almost prodigal in endowing us with nearly 10*ma* of convergence,—that is to say, in permitting us to converge for a distance at which we never work. No such prodigality has been shown, however. In this case, as in the others, we have received only what is strictly necessary : the *punctum proximum* represents, indeed, the maximum of convergence of which an individual is capable. Now, in order to continue any muscular effort for a long time, it is essential that this effort do not require from the outset all the force that is at its disposal.

No eye maintains for more than an instant vision at the distance of its nearest point of accommodation, for the very reason that vision at that distance requires the maximum contraction of its ciliary muscle. In the same way, nobody can maintain convergence at its maximum without crossed diplopia immediately showing him that his power of adduction is exhausted. The mere mention of these facts suffices to prove that the position of the *punctum proximum* of convergence does not correspond to the distance at which the individual can work, nor the maximum of convergence the power which would be at his absolute disposal during the continuance of his occupation. Of this force he can make use of only a *relative* quantity, while he must have a certain amount *in reserve* to replace the force expended.

It is of primary importance to be acquainted with the relations between the quantity at disposal and the totality of this function, to know, there being given, on the one hand, the distance at which a person wishes to work, and, on the other, his power of convergence, whether or not the latter is sufficient.

We have tried to determine this *quota of convergence,*[1] and our experiments seem to demonstrate that this reserve amount ought to be about twice as great as the convergence required by the work.[2]

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[1] Landolt, Arch. d'Ophthalmologie, March, 1886, Insufficiency of the Power of Convergence; The Ophthalmic Review, v., 1886; Bericht der Ophthalmologischen Gesellschaft, Heidelberg, 1886.

[2] It is interesting to note the difference which exists, in this regard, between convergence and accommodation. We have found, indeed, that one-third of the amplitude of accommodation suffices for the reserve, while two-thirds of the amplitude are necessary for the reserve part of convergence.

It is more than fifteen years since we undertook our investigations concerning the reserve amounts of convergence and accommodation. Their results seem mutually confirmatory. It must not be forgotten, however, that conclusions from experiments with muscles generally, and with the ocular muscles particularly, may be somewhat uncertain.

Thus, in order to work at the usual distance of $\frac{1m}{3}$, that is to say, with $3ma$ of convergence, $2 \times 3 = 6ma$ should remain in reserve. In other words, the individual should possess at least $3 + 6 = 9ma$ altogether. Hence we have here just the $9ma$ that we found at the outset. They thus represent the amount which is strictly necessary for usual work.

It is true that in this case, as in others, there are people who are much more fortunate, privileged ones who have at their disposal a much greater convergence capital (and who do not even always use it). But along with them, there are also the unfortunate whose convergence is not sufficient even to accomplish the daily work,—indeed, does not even allow them to use their eyes in any way without fatigue.

We designate this condition as *insufficiency of convergence.*

It is easy to see that, in spite of the normal amplitude, convergence may be insufficient for exceptionally near work. Thus, in order to work at 25 centimetres, or $\frac{1m}{4}$,—that is to say, with $4ma$,—$3 \times 4 = 12ma$ of positive convergence are requisite. He who possesses only $9ma$ will evidently be incapable of doing this work binocularly, and will feel the phenomena of fatigue which characterize insufficiency of convergence. But in such a case the insufficiency is only relative; it is not the symptom of a morbid condition, although claiming at least the optical aids that we use at times to remedy pathological insufficiency.

Insufficiency of convergence properly so called is characterized by a diminution of the positive part of the amplitude of convergence. The *punctum proximum* is farther away, and convergence for greater or less distances is difficult and gives rise to those symptoms of *asthenopia* which are encountered in the early stages of divergent strabismus,—viz., fatigue of the eyes, crossed diplopia, pains in the head having their seat especially in the forehead, vertigo, and general *malaise.*

When one examines, as is indispensable, not only the positive but also the negative part of the amplitude of convergence, it is found that this function may be altered in various ways, and differently at its two extremities.

Thus, as the line *B* of our diagram (Fig. 48) shows, the total amplitude may have a normal value of $10ma$, and yet there may exist an insufficiency of convergence, the maximum of convergence being only $7ma$. The two metre-angles which are wanting in the positive part are, so to speak, carried over to the negative: divergence is increased by the same amount that convergence, properly so called, is diminished. The entire amplitude has passed over towards the negative side.

In other cases (*C*, Fig. 48) the diminution of convergence is more marked than is the increase in divergence.

Sometimes the latter is normal (*D*), and the former alone is altered.—In *E*, the opposite is the case.

At times, also, convergence and divergence are both restricted ($F$, $G$, $K$).

It may even happen, as in $I$, that the whole amplitude of convergence is negative. The individual can no longer converge, he cannot even give his lines of sight a parallel direction. He is subject to divergent strabismus. By bringing into action his entire adductive power, he succeeds, at the very utmost, in not letting his eyes diverge more than half a metre-angle. On the other hand, he can increase his divergence even to $3ma$. Hence he possesses a certain amplitude of convergence: it amounts to $2.5ma$; but its whole extent is negative. This "convergence" is therefore only a less or greater divergence.

Insufficiency of convergence has very different *causes*. It may, in the first place, be the consequence of a material lesion of the brain or of the spinal cord. Thus Parinaud[1] has found it in a case of *neoplasm* having its seat at the outer edge of the right cerebral peduncle and extending to the cerebellar peduncle, to the fourth ventricle and to the aqueduct of Sylvius, where it affected the nucleus of origin of the third pair. The same author reports, in the same place, a case of *insular sclerosis* in which, as he says, "*le mouvement de convergence se faisait très incomplétement au delà de* 30 *cm., la vision associée n'existe plus, elle développe du strabisme, et l'on constate tous les signes d'une insuffisance prononcée des droits internes, en couvrant alternativement chaque œil.*"[2]

In 1885,[3] along with Dr. Huebscher, then my *chef de clinique*, I observed a case of *tabes dorsalis* in which convergence was considerably limited. This case is reported with others in a monograph on paralysis of convergence published by another of our students, Dr. Borel,[4] in the *Archives d'Ophtalmologie*. Similar cases have been published by de Watteville,[5] while Granger Stewart[6] has seen a crossed diplopia produced during the movement of convergence in the case of an ataxic. Gowers,[7] Samelsohn, and Stölting and Bruns[8] likewise mention the loss of the convergence movement in tabes.

Since these publications attracted the attention of physicians to the insufficiency of convergence in locomotor ataxia, observations of this kind have become very frequent.

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[1] Parinaud, loc. cit., p. 162, and Soc. française d'Opht., 1886, p. 23.

[2] Parinaud, Arch. de Neurologie, 1883, p. 162. See also Soc. française d'Opht., 1886.

[3] Landolt, Die Insufficienz des Convergenzvermoegens, Heidelberg. Ophth. Gesellschaft, 1885; Soc. franç. d'Opht., 1886, and Ophthalmic Review, v., July and August, 1886; Traité complet d'Opht., iii. p. 923; Refraction and Accommodation, p. 504; Stevens, Functional Nervous Diseases, New York, 1887, Archives of Ophthalmology, xv., 1887.

[4] Borel, Paralysie de la convergence dans l'ataxie locomotrice, Arch. d'Opht., Novembre, 1887.

[5] A. de Watteville, Ueber die Laehmung des Convergenzbewegung des Auges im Beginne des Tabes Dorsalis, Neurolog. Centralblatt, No. 10, 1887.

[6] Granger Stewart, Eye Symptoms in Locomotor Ataxia, Brain, ii.

[7] Gowers, Diseases of the Nervous System, i. p. 298.

[8] Stölting u. Bruns, Graefe's Archiv, xxxiv., 3, p. 92.

In fact, it is easy to understand that the affections of the nervous system, whatever their nature may be, can produce alterations of this kind. After simple insufficiency of convergence they may even bring about the complete abolition of convergence.

A somewhat different form of insufficiency of convergence is that which accompanies the *neuroses*, and in particular what is called *neurasthenia*. We have applied to it the adjective *neuropathic*.[1]

Here again the excursions of the eyes may be normal; the synergy of the abductors (divergence) is at times normal (*D*, Fig. 45), or it may even be augmented (*C*); only the adduction has suffered. One often finds, however, in such cases, that the range of convergence is reduced at both ends, as is shown at *F* and *G* of Fig. 45.

In this category must be placed the insufficiency of convergence observed in *exophthalmic goitre*,[2] *chronic alcoholism*, *hysteria*,[3] and *neurasthenia*.[4]

Let us recall our observation reported in the paragraph concerning the associated paralyses, wherein an almost absolute insufficiency of convergence accompanied, in the case of a hysterical woman, abolition of the power to look up or down.

Neurasthenia, while it is particularly frequent among women, nevertheless also attacks the sex which merits so little the name of the "strong." It often has as its cause intellectual or physical strain, excess of work, of care, and also excess of pleasure. In this case, one is often much surprised to find a man of splendid figure and remarkable strength, apparently quite healthy, gifted with two perfect eyes, but yet incapable of using them, as a severe asthenopia manifests itself whenever he tries to do any reading, or work of whatever kind at a short distance.

I have met many cases of this sort, especially in my American *clientèle*, and I find an explanation of them in the ardor which, in that country of illimitable resources, characterizes the struggle, not only for life, but for fortune and what it brings.

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[1] Landolt, Ophth. Gesellschaft, Heidelberg, 1885. This insufficiency of the innervation of the adductors, or, briefly stated, of convergence, constitutes a phenomenon analogous to the insufficiency of the orbicularis that Rosenbach has pointed out and Bannas (Thesis, Breslau, 1893) has confirmed as a characteristic symptom of neurasthenia.

[2] P. L. Moebius, Ueber Insufficienz der Convergenz bei Morbus Basedowii, Centralblatt für Nervenheilkunde, 1886, p. 356.

[3] Borel, Affections hystériques des Muscles oculaires, Arch. d'Opht., 1887, p. 356.

[4] Compare, among others,—

Henry D. Noyes, Diseases of the Eye, p. 88.

C. Stedman Bull, in Soelberg-Wells, Diseases of the Eye, p. 719, fourth American edition.

Th. R. Pooley, New York Medical Journal, 43, p. 179.

R. J. McKay, American Journal of the Medical Sciences, October, 1882.

C. M. Culver, Convergence Anomalies, Albany Medical Annals, viii. p. 137, 1887.

D. B. St. John Roosa, Medical Record, New York, p. 429, 1890.

K. Cross, Bristol Med.-Chir. Journal, 1893, p. 73.

De Lapersonne, Recueil d'Opht.

At times, also, this weakness of the adductive power accompanies *anæmia*, or it remains as a vestige of some *debilitating disease*,—typhoid fever, influenza, difficult accouchements, hemorrhages, etc.

If this form of asthenopia can strictly still be called *central*, because it finds its explanation in weakness or in a lack of energy of the centre of innervation of convergence, we may hesitate to apply the same name to a *third* form of insufficiency of convergence, which is not rare, and which is essentially due to a *lack of use.*

The type of this form of insufficiency is represented by myopes, of whom we have already spoken, who possess good eyes so far as their visual power as well as their motility is concerned, but which diverge as soon as the possibility of fusion is abolished, whether by the exclusion of one eye, by a vertical prism, or by any other means.

With them convergence is weak, rudimentary, insufficient, because one of the stimulants which cause the emmetrope or hyperope to converge, even when there is no fusion,—that is to say, the accommodation,—is lacking.

This is clearly proved by our measurements of the amplitude of convergence.[1] These show that even myopes whose binocular vision is intact, frequently have an amplitude of convergence which is inferior to that of emmetropes; the *punctum proximum* is farther away than in the case of hyperopes or emmetropes, and, although abduction is often augmented in myopes, adduction is almost always restricted. If there be added still another circumstance which interferes with binocular vision, we need not be astonished if convergence is not normally developed in such cases.

That which is the rule with myopes whose myopia is of a certain amount may, as we have demonstrated, happen to eyes of any other kind of refraction, if the convergence is not often or not energetically exercised, in spite of an otherwise satisfactory development of the muscular apparatus of the eyes.

We see that this form of insufficiency of convergence is essentially due to a *lack of exercise.* It is, in our opinion, not comparable to the insufficiency resulting from a central lesion; it is in no wise a paralysis.

Let us suppose the case of a vigorous man with good legs. He is put on a horse. While at a walk, he sits well enough in the saddle, but as soon as the horse trots he wabbles and loses his balance. We recommend him to cling with his legs; he does his best, but at the first turn, at the least acceleration of the pace, at the least shy of the horse, he falls. Do we commiserate him, explaining to him that he is the victim of a cerebral affection, of a lesion of the centre of innervation of his adductors? Not at all. Though we know that the adductor muscles do not contract without innervation, and that their centre of innervation is in the brain, we content ourselves with saying simply that he is not in the habit of using the ad-

[1] Landolt, in Ellaby, Paris Thesis, p. 68, 1884, and Amplitude de Convergence, Arch. d'Opht., March, 1886.

ductors of his legs. We encourage him, convinced that, with practice, his muscles, *as well as their innervation,* will be strengthened, and that he will finally be able to *cling with his legs* as much as is necessary.

The same thing happens in the case of insufficiency of convergence of our third class; convergence is defective because it is not exercised. But we do not think that it can be counted in the class of the insufficiencies of *central origin,* for this term is generally understood to imply a material cerebral lesion.

Finally, the *fourth* form of insufficiency of convergence is represented by cases wherein the internal recti muscles are really weak, in which the fields of fixation show a limitation at the nasal side. It is the *muscular insufficiency* which gives rise to muscular asthenopia when binocular vision exists, but to divergent strabismus when it does not exist.

The existence of this muscular insufficiency has been denied,[1] as if the ocular muscles alone among the muscles of the human body could neither be nor become insufficient for the work which they have to accomplish, however hard that work may be. If, instead of making theoretical systems, trouble were taken to thoroughly examine patients, notably their fields of fixation, such a statement would not be made. Even the fatigue resulting from convergence which has been too long maintained ought of itself to be able to bring about an insufficiency, at least a transient insufficiency, of the muscles which cause convergence.

On the other hand, we see that in cases where convergence is little used, not only does this function remain or become rudimentary, but the muscles also become weak. Thus it is that with myopes the fields of fixation are very often limited at the nasal side. This might occasion surprise if one thought that, although not contracting much for convergence, the internal recti are nevertheless exercised in the associated lateral movements. But we have already seen that the usual lateral excursions are of only a few degrees, and are not comparable, either as to extent or as to duration, with the exercise to which these muscles are subjected during convergence, in the interest of binocular vision. In this form the insufficiency of the adductor muscles is *secondary.*

But is there not also a primary muscular insufficiency, due to a congenital defect of development of one or even of a group of ocular muscles? Such an anomaly is met in other parts of the body; why should the eyes be exempt from it?

Fuchs has already demonstrated in a remarkable monograph[2] certain differences which may exist in the disposition of the ocular muscles. Le Double has published in the *Archives d'Ophtalmologie* another work which is still more conclusive in this respect.[3]

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[1] See, among others, International Congress of Medical Sciences, Berlin, 1890, section of ophthalmology.

[2] Fuchs, Arch. f. Opht., xxx., 4, S. 1, 1884.

[3] Le Double, Arch. d'Opht., p. 218, 1894.

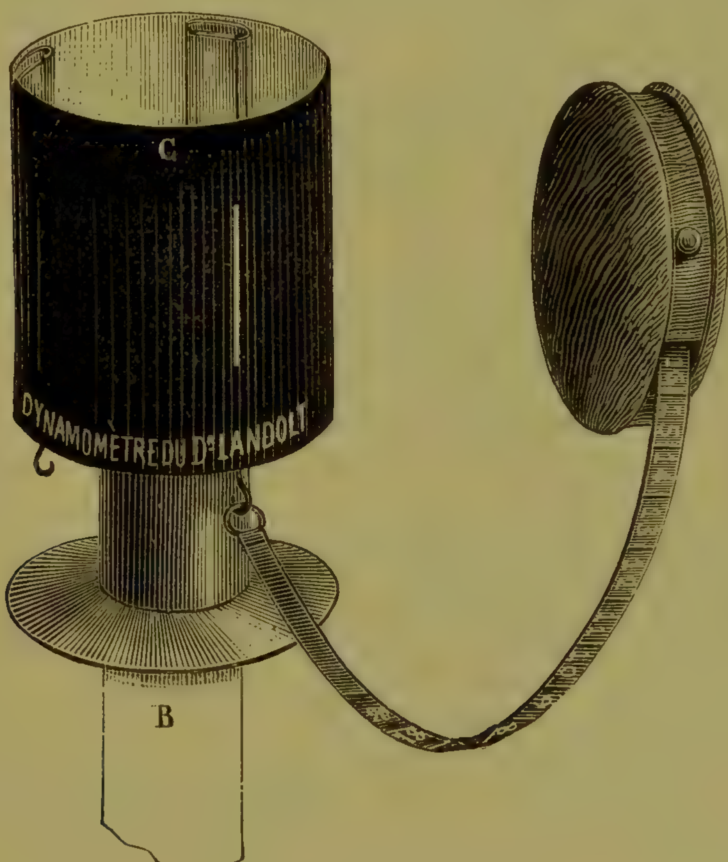
Hence we may admit, in addition to insufficiency of convergence due to *central lesion*, to *neurasthenia*, or to *lack of exercise*, one due to *defective development of the adductor muscles.*

## DIAGNOSIS OF INSUFFICIENCY OF CONVERGENCE.

Since the time of the illustrious von Graefe, many more or less complicated procedures have been invented for the examination of convergence and its anomalies.

Thus it has been proposed to measure the converging power of an individual by making him fix first a distant object, and then a near one through adducting prisms. The strongest prism that he can tolerate without seeing the object double is supposed to measure his power of convergence.

FIG. 49.




Landolt's ophthalmodynamometer.

It has also been asserted that the best means of estimating the convergence power of an individual is to measure his power of divergence.[1]

To us, however, it has always seemed that, if one wishes to know a person's power of convergence, the simplest and the most logical method is to determine the distance of the nearest point to which he can converge.[2]

In order to know whether convergence is normal or not, the value thus

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[1] Mannhardt, Arch. f. Ophth., xvii., 1871.

[2] Landolt, Soc. française d'Opht., 1883; Ophthalmic Review, 1886, p. 203. Refraction and Accommodation, p. 283.

found has to be compared with the value found in the same way in healthy persons.

The instrument that we have devised for the estimation of the power of convergence (as well as that of the accommodation) is known by the name of OPHTHALMODYNAMOMETER. It consists of a cylinder (*C*, Fig. 49) blackened on the outside, which can be fitted on a candle (*B*) of ordinary size. The cylinder has a vertical slit about a third of a millimetre in breadth (Fig. 49), a series of fine openings which form together a vertical line, and a circular aperture about one millimetre in diameter. The slit and the openings are all covered with ground glass. When the candle is lighted, they constitute luminous objects of fixation. Beneath each opening can be attached one end of a tape measure that is rolled up by means of a spring. The tape is graduated on one side in centimetres, on the other in the corresponding numbers of metre-angles (or, what amounts to the same thing, in dioptries).

To determine the *maximum of convergence*, we use the luminous slit as the object of fixation. The tape measure is drawn out to about seventy centimetres, its case being held beside one of the eyes of the patient (theoretically, on a level with its centre of rotation), while the object of fixation is placed in the *median line*. If the patient sees the object single, then, by pressing on the knob of the case, the spring is made to roll up the tape, and thus the observer brings the fixation-object nearer to the eyes, taking care, however, that it always remains in the median line. So soon as the person under observation begins to see double, the *near point of convergence* is attained. In fact, when the eyes have no longer the power required to fix the object simultaneously, there exists a divergence relatively to its position, and, consequently, crossed diplopia.

At this instant, one side of the tape gives in *centimetres* the distance of the *punctum proximum* of convergence, and the other side the corresponding *maximum of convergence* in *metre-angles*. For instance, eleven centimetres correspond to nine *metre-angles*.

As the motor apparatus of the eyes does not always perform its functions with as great accuracy as is desirable, it is necessary to repeat the experiment several times at one sitting, and, especially in pathological cases, on different days.

It is furthermore important, during all the time that the object is being brought nearer, to encourage the patient energetically to fix it. It will also be observed that the *punctum proximum* of convergence, when thus obtained, lies nearer to the eyes than when the object is first placed within this distance and then withdrawn from the patient. By the latter method accurate results are not obtained. Hence we advise that the object of fixation be brought *toward* the patient from a point for which he can easily converge.

Here, as in all investigations concerning binocular vision, it may happen that the patient is not certain at first whether he sees double or not. In

cases, for instance, where one eye is considerably weaker than the other, the patient easily overlooks the retinal image of the former, so that he does not see double, notwithstanding a faulty direction of the eyes. The same thing can occur when, from other causes, perfect binocular vision has never existed, —as, for instance, in high degrees of myopia with almost similar eyes, but so short-sighted that perfect binocular vision is impossible without artificial aid. Even individuals with healthy eyes, but with ill-developed intellectual faculties, are frequently unable to say what or how they see. In such cases the examination can be rendered much easier by holding a colored glass before one eye until the patient is aware of the double images. In the majority of cases it is sufficient to have made the patient perceive the second image by means of a red glass. We therefore use it only at the commencement of the experiment, and sometimes to verify the patient's answers.

It is always advisable to watch the eyes of the person under examination: in most cases they at first follow the object correctly, then at a certain distance one eye seems to stand still, as if it hesitated, and finally, when the object has approached still nearer, it turns outward.

At this last moment, however, the *punctum proximum* has already been passed over. The maximum of convergence is attained when the hesitation and crossed diplopia begin.

To define the range of *accommodation*, the fine openings of the dynamometer are used. These are gradually brought nearer the patient till they appear *indistinct;* the result is read off on the tape in dioptries instead of metre-angles, and the maximum of refraction in the place of maximum of convergence. When a person is emmetropic, the maximum of refraction of which he is capable is equal to the range of accommodation.

The same line of luminous points may be used in investigating the *relation existing between the converging*, or motor, and the *accommodative*, or optical *apparatus* of the eyes. When convergence and accommodation harmonize, the patient will see single and distinctly the row of luminous points as such. If convergence be at fault, the line will appear double, the diplopia being crossed if there be insufficiency, or homonymous if there be excess. Should there be a failure in the optical adaptation, the points will appear blurred.

The circular aperture of the dynamometer is useful in the analysis of all kinds of derangements of motility which are accompanied by diplopia.

The case of the dynamometer also contains a little frame, with a handle, in which threads, hairs, small objects, such as printed letters, or a diaphragm with fine holes, can be placed to define the range of accommodation.

It has been said above that the maximum convergence is on the average $9ma$ to $10ma$. As a matter of fact, we have rarely met insufficiency of this function among persons capable of converging to a point ten centimetres distant.

Such a case, however, might be present if the individual were obliged to work binocularly at a particularly short distance. Indeed, since, according to our experience, two-thirds of the convergence must be kept in reserve and only one-third ought to be expended, the $9ma$ would suffice, theoretically, only for a distance of thirty-three centimetres. Twenty-five centimetres $\left(\frac{1m}{4}\right)$ would demand $8ma$ in reserve, hence $12ma$ altogether.

It is not rare to find this amount of converging power,—indeed, even more, especially in the case of persons who have trained themselves to it,—but any one who possesses only 10*ma* as the maximum of convergence would soon tire himself out by working at a distance of a fourth of a metre.

On the other hand, 8*ma* of maximum convergence would not constitute of itself an absolute insufficiency of convergence. The individual need only move farther from him the object on which he is working, or frequently interrupt his work for the purpose of resting his muscles, and thus restore their exhausted power. But 8*ma* seems to us a minimum below which convergence could scarcely fall without symptoms of asthenopia showing themselves. Under such circumstances the patient says that the first moments of work pass fairly well, but there soon comes a feeling of fatigue, more or less localized, accompanied by pain in the forehead, the temples, or the head generally. At the same time, vision becomes indistinct, from a cause which escapes most patients. However, those among them who have some ability as observers remark that the object of fixation becomes double; for instance, they see the printed page separate itself into two images, which glide in opposite directions, one of the images undergoing the apparent displacement, while the other continues stationary. At this moment, they have at times the distinct sensation that one of the eyes has deviated outward.

A phenomenon which is frequently noticed by persons with insufficiency of convergence is a certain difficulty in successively directing the gaze from one to another of different near points whose distances vary. These different successive fixations are not executed with the promptness and precision habitual to healthy eyes; the objects, before being fixed, appear indistinct or double, and the movements of the eyes, of which normal eyes are not conscious, occasion a certain feeling of annoyance.

The momentary occlusion of the eyes, or a more or less prolonged gaze into space, dissipates these symptoms, and fixation again becomes possible for a certain time; but the fatigue is renewed, becomes more intense, and necessitates a new interval of repose. For a certain length of time it always causes marked fatigue.

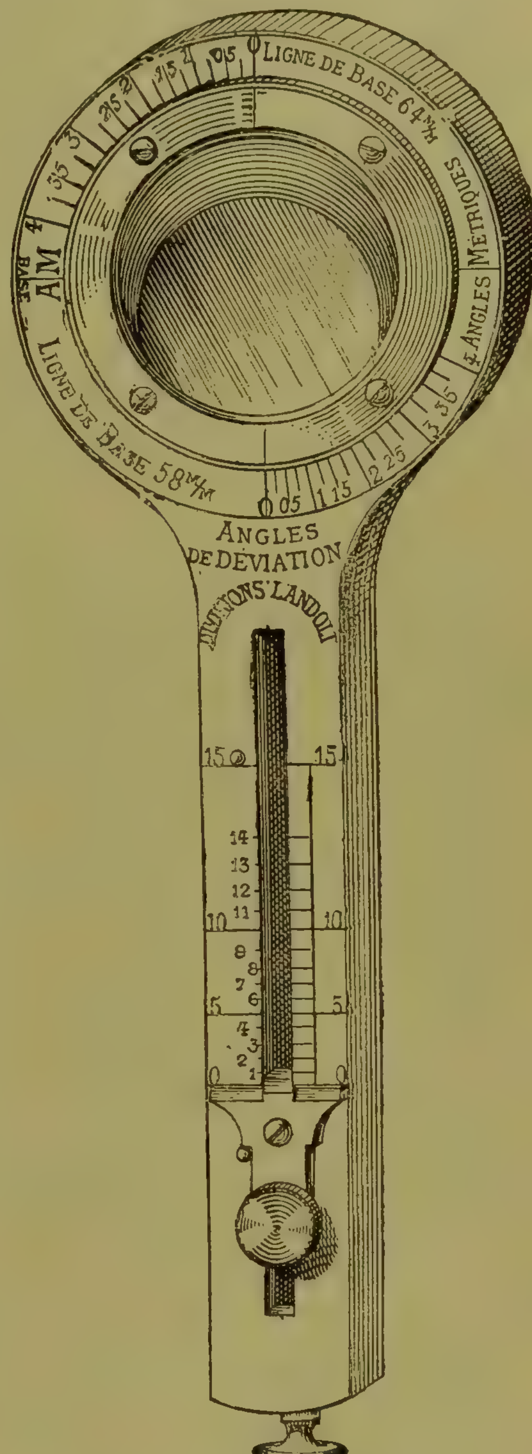
In other cases, asthenopia increases to such a degree as to render all work impossible, or to provoke, at the end of a short time, intense cephalalgia, hemicrania, with all the concomitant train of symptoms, such as nausea, vertigo, etc.

A short objective examination sometimes suffices to recognize insufficiency of convergence. It is enough to make the patient look at an object, which is brought gradually nearer to his eyes, along the median line. It can be seen that at a moderate distance the movements of convergence are relaxed and become undecided. The patient has a tendency to recoil, to withdraw from the object of fixation. If the latter be brought still nearer the eyes, they commence to show oscillations, and at length entirely renounce fixation and become divergent. This phenomenon manifests itself,

of course, only in one of them, which suddenly stops, then executes an associated movement outward, while its companion continues to turn inward.

This is, after all, the same phenomenon that occurs, under normal circumstances, as soon as the object of fixation is brought nearer than the *punctum proximum* of convergence; only in the pathological condition it is produced earlier; indeed, even sometimes during vision at great distance, when the maximum of convergence is zero or negative (*I*, Fig. 48).

Fig. 50.




Herschel's double prism with Landolt's divisions.

The insufficiency being thus recognized, and the maximum of convergence determined by means of the dynamometer, we measure also the negative part of this function by means of abducting prisms, or, more simply, with Herschel's double prism (Fig. 50).[1]

This instrument, which the optician Crétès, of Paris, formerly made for the use of ophthalmologists, consists of two prisms of equal power which turn around the same axis in opposite directions. When the apices of the two are directed in exactly opposite directions, they neutralize each other, their surfaces being parallel. On the contrary, when the apices have the same direction their action is the sum of the powers of both prisms.

From one of these positions to the other the double prism presents, therefore, all the degrees, from zero to the sum of the two prisms of which it is composed.

The degrees of the resultant prism are engraved on the handle of the instrument, and are indicated by the position of the knob, by pressure on which the prisms are moved.

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[1] A convenient arrangement for this purpose, devised by Noyes (Diseases of the Eye, p. 193), is a series of square prisms set one above another in a frame and increasing in power by intervals of one degree of deviation. One series may have odd numbers up to nine degrees, and another even numbers up to eight degrees.

We have had substituted for these figures, which formerly indicated the angles of opening of the prisms, the more rational ones corresponding to the *angle of deviation* produced by the combined prisms.

On the circular mounting of the instrument, we have had engraved the corresponding *metre-angles* for a base-line of fifty-eight millimetres (children), and for a base-line of sixty-four millimetres (adults).

Nothing is easier, then, than to find the maximum of divergence and express it in metre-angles. One need only make the patient fix a distant candle-flame and turn before one of his eyes the prism held in such a way that the apex of the resultant prism shall be directed towards the temple of the same side. As soon as a homonymous diplopia commences to manifest itself, we know that we have reached the limit of divergence (—r), and we read the degree of it on the mounting of the instrument.

By subtracting this amount from that of the maximum of convergence (p), we obtain the amplitude of this function,

$$a = p - r.$$

If acquaintance with the minimum of convergence be interesting even with reference to the nature of the insufficiency, it is still more so, as we shall see, when regarded from a therapeutic point of view.

In order to complete our diagnosis and learn whether the power of convergence alone has changed, or whether there exists a weakness of the muscles themselves, we must examine the *excursions of the eyes* by means of the perimeter, according to the method which we have formerly described.

Finally, the examination of the state of motility may be completed by the determination of the so-called position of equilibrium of the eyes.

The term *position of equilibrium* is used to designate the direction taken by the eyes when in a state of minimum innervation or of *absolute repose.*

Nothing is more difficult than to find this position. In fact, it could not be determined by any other means than by some subjective method,—that is to say, by the aid of the indications given by the person himself under examination. For it is not the direction of the pupillary axes that is to be sought, but that of the visual lines, concerning which the patient is the sole judge. But any object of observation, whether near or distant, influences the direction of the two eyes, even when it is seen only by one of them, and that not only by the effort of accommodation which the vision of this object demands, but even by the distance at which the individual supposes the object to be, whether correctly or fallaciously.

The eyes are so mobile, and the innervation of their motor apparatus is both so complex and so delicate, that even a semi-conscious thought suffices to modify their relative direction. It is modified even during dreams. Only profound narcosis or death brings about the absolute relaxation of the ocular muscles. But under such circumstances it might be difficult to get an answer from the person consenting to undergo the experiment.

In default of this absolute resolution of innervation, one seeks at least to withdraw the patient as much as possible from the influences capable of modifying the direction of his eyes. When the vision is concerned with very distant objects, the least possible demand is made upon the accommodation. On the other hand, the tendency to fusion is diminished by rendering dissimilar the images of the two eyes by means of a colored glass, and even more so by means of a prism which produces insurmountable vertical diplopia.

In conducting this investigation, Stilling[1] makes the person under examination close the eyes and then say whether a star or some other distant luminous object appears single or double at the moment when the eyes are opened. Or, again, he makes the patient look at this luminous point and then successively covers and uncovers one of the eyes to learn if, in spite of the suppression of binocular vision, the excluded eye remains in parallelism with the other, or if it modifies its direction by converging, by diverging, by upward or by downward deflection.

Although the author has found in certain cases a homonymous diplopia by means of this proceeding, the notion of the great distance of the object fixed certainly suffices in other cases to prevent the eyes from converging under these circumstances. On the other hand, there must be also many persons who do not diverge, because no object which is fixed under ordinary conditions requires this direction of the visual lines.

A method which Donders used to determine the condition of rest of the eyes appears to us preferable to that which we have just mentioned. The Dutch physiologist brought the person to be examined into an absolutely dark room. One of the eyes was covered with a red glass and the patient's face directed towards one of the walls of the apartment. At the end of a certain period of repose, an electric spark appeared before the patient. Single vision or the nature of the diplopia in which this light appeared indicated the direction which the eyes had taken during the absence of any object of fixation. This experiment has the advantage that the rapidity with which the luminous object appears and disappears does not permit the patient to modify the direction of his eyes according to the distance which he attributes to the object.

Snellen uses in his clinic at Utrecht a very ingenious method for determining the degree of divergence or convergence of the eyes in what is at least a relative state of repose. This experiment also is made in an absolutely dark room. The patient has in front of him a great number of apertures made in a board and arranged as a horizontal line, covered with a *red* glass, and illuminated from behind; below one of these holes is another covered with a *green* glass.

The person under examination wears spectacles of which one glass is red, complementary to the green of the aperture of the board, and the other

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[1] Stilling, Arch. f. Augenheilk., xv., 1, S. 73, 1885.

green, complementary to the red covering of the line of apertures in the board. Hence the person sees with one eye only the red points, while the other perceives only the green point. Then the patient is asked to say whereabouts along the line of red points he sees the green one. The distance between the place where the green point actually is and the place where the patient supposes it to be is equal to the tangent of the angle of deviation. The latter can be read directly on the scale.[1]

A good method of determining the position of equilibrium or the latent deviation of the eyes is that of Maddox, which, under the name of *rod-test*,[2] is pretty generally known and appreciated.

That author places before one of the eyes of the person to be examined a glass rod which is not more than two or three millimetres in diameter. Any luminous point looked at through this cylinder is changed into a long line of diffusion. The direction of this line is necessarily perpendicular to that of the rod. When looking thus towards a distant candle-flame, we receive from this object two entirely dissimilar images, on one side the normal one, and on the other the luminous line. The difference between this line and the image received by the free eye is still greater when one of them is reddened either by using a red rod or by covering one of the eyes with a glass of this color. It is evident that the rod must be opposite the pupil. To facilitate this position, Maddox now uses a series of parallel rods, so that the person under examination is always sure to look through at least one of them. Such a series of rods is mounted on a disk of metal of a size to fit into any ordinary trial frame.

If it be a question of determining horizontal deviations, the rods are given an exactly horizontal direction, and the light appears to the eye before which the rods are placed, in the form of a vertical line. If the eyes are normally directed, this line passes exactly through the candle-flame. Convergence will provoke homonymous diplopia, divergence crossed diplopia. For vertical deviations the rods are held vertically, so as to produce a horizontal line of light.

The *degree* of this deviation may be measured in different ways. Thus one may find a prism which produces fusion of the two images. If one uses for this purpose a double prism which has our graduation, one may obtain the degree of deviation, expressed at once in ordinary angles and in metre-angles.

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[1] M. J. Reboud has used Snellen's method in investigations concerning the direction that the eyes take under the influence of what he calls *l'habitude physiologique* (physiological habit).

He finds that this direction is, in the majority of cases, parallelism or divergence (sixty-one per cent.). Convergence (thirty-nine per cent.) is met with when an effort of accommodation is necessary in order to procure for the fixing eye distinct retinal images.

It is evident that the results of these investigations constitute further confirmation of Donders's law concerning the relation between convergence and accommodation.

[2] E. Maddox, A New Test for Heterophoria, Ophthalmic Review, Mry, 1890.

This deviation may also be measured by means of the mural division which I described many years ago, and have mentioned above.[1]

The different methods which Dr. George T. Stevens uses for the determination of what he calls heterophoria are to be found in the article on the Estimation of the Balance of the Extra-Ocular Muscles, vol. ii. of the present work.

Professor Straub,[2] of Amsterdam, recommends the following method for the determination of the position of repose of the eyes during vision at different distances:

A ribbon, a metre and a half long, has at one of its ends an oblong mirror, and at the other end a wooden plate, large enough to cover one eye. The person under examination is seated with his back towards the window; the observer, sitting in front of him, holds the mirror horizontally in front of his own forehead and tells the patient to fix the image of some object reflected in the mirror. By rapidly and alternately covering the eyes of the patient, he ascertains if they have a tendency to be dissociated, and in what direction.

Priestley Smith[3] very ingeniously remarks that the corneal reflex from an ophthalmoscope may serve to teach us the direction which the eyes have when deprived of binocular vision. Indeed, the ophthalmoscope mirror presents a luminous object of fixation to only one of the eyes of the person examined. Hence the other can, without prejudice, take the direction which is easiest for it,—a direction which its corneal reflex then reveals to the observer.

Instead of diminishing the tendency to fusion of the images belonging to the two eyes by making them dissimilar, as Maddox, Stevens, Snellen, and others do, this fusion may be rendered impossible by the aid of a very simple method devised by von Graefe. This consists in placing before one of the eyes a vertical prism. The person examined then sees, for instance, a candle-flame with a vertical diplopia which he cannot overcome; he is thus brought to abandon his eyes to their direction of relative repose. If this direction be parallelism, the two images will appear to be in the same vertical line. In addition to the difference in height between the two images, convergence will show itself by homonymous diplopia, divergence by crossed diplopia. The degree of these deviations may be measured by means of our tangential division, or by the prism which brings the two images into the same vertical line.[4]

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[1] Landolt, Strabométrie, Ann. d'Ocul., 1875.

[2] Straub, An instrument for the determination of convergence power and the position of rest of the eyes, Ophthalmic Review, April, 1892.

[3] Smith, On the corneal reflex as a test of fixation and deviation, Ophthalmic Review, February, 1892.

[4] See also J. F. Herbert, A scale for the speedy and accurate determination of the anomalies of the ocular muscles, Ophthalmic Record, p. 324, 1894. The author uses a scale which is not divided into angles, but into *prism-dioptries*, according to the proposal of C. F. Prentice.

Used in this way,—that is to say, with the fixation-object at a great distance,—this method of our eminent master may help to elucidate the complex problem of the motility of the eyes. We dare not say as much concerning its application for short distance, for which von Graefe used it, in determining what he called insufficiency of the internal recti muscles.[1]

This method, which the great Berlin ophthalmologist called "*Gleichgewichtsversuch*," is, however, still so much in use that we cannot pass it in silence. It is employed at the distance of near-work,—*i e.*, at about thirty centimetres.

In front of one of the eyes is placed a vertical prism of about six degrees, which produces a vertical diplopia of the object of fixation. This object consists of a black point through which passes a vertical line. If the two vertical lines do not join, but are separated from each other, the horizontal prism which superposes one on the other is sought.

In the case of *crossed* diplopia, the prism thus found ought, according to von Graefe, to give the amount of insufficiency of *convergence*.

Von Graefe attached, moreover, great importance to the measurement of what he called the abducting power, or simply *abduction*, at working distance, as well as at a distance of several metres.

Abduction at a great distance is nothing else than potential divergence of the lines of sight, or what we call the *negative portion of the amplitude of convergence*.

To the same extent that investigation of absolute divergence is rational and useful, so is the opposite attempt (by means of adducting prisms) devoid of sense, utility, and accuracy.

The criticism of this proceeding is to be found in our work on the movements of the eyes and their anomalies.[2] We shall not renew its discussion now ; it is easy to see that von Graefe's investigation could not give useful results. By obliging the person examined to change his convergence (or divergence) without changing his accommodation,—since the distance of the object remained the same,—he placed him in conditions of vision which were entirely abnormal.

However interesting these investigations concerning the *position of equilibrium* and *latent deviations* of the eyes may be, they in no wise suffice to inform us either of the aptitude of the eyes for work or of the nature of the asthenopia.

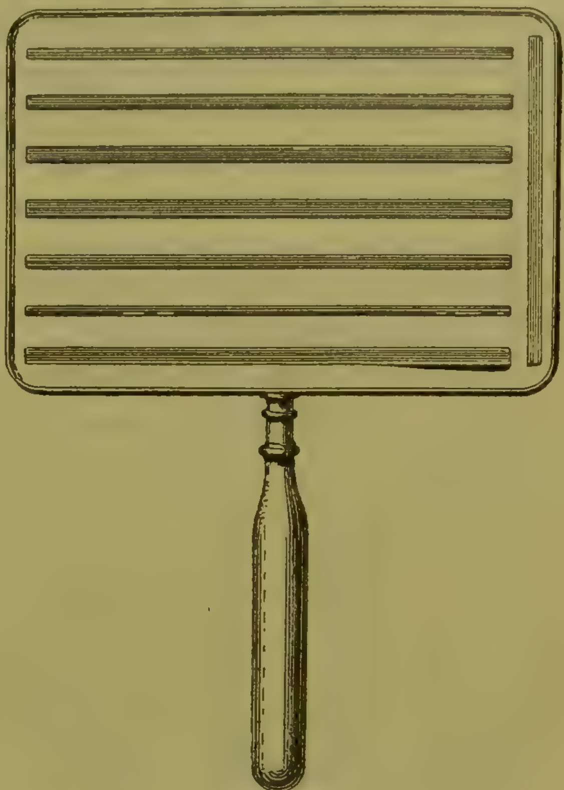
It would be quite wrong, for instance, to conclude, from the existence of what has been called "latent divergence," that insufficiency of convergence was present. Eyes in the condition of absolute repose may diverge, the equilibrium test may indicate a distinct crossed diplopia, while nevertheless the power of convergence may be perfect. Study of the position of equilibrium teaches us, at the utmost, the tendency of the eyes to diverge

[1] Von Graefe, Arch. f. Ophth., viii. 2, 1862.
[2] De Wecker et Landolt, Traité complet d'Opht , iii. pp. 912–915.

or converge, the direction which they would probably take if binocular vision were lost. It is, like the measurement of abduction, a guide in our therapeutic undertakings.

A more instructive test is to observe the eyes during fixation, and especially during near-work. We have devised for this purpose a most simple instrument.[1] It consists of a glass plate, framed, and mounted on a handle. (Fig. 51.)

Fig. 51.



Landolt's kinophthalmoscope.

On this glass we fix bits of print or figures of all sorts. We hold it at the desired distance between our eyes and the patient's, and, while he fixes, reads, or follows the test-objects, we observe his eyes through the glass. This instrument may be called a "kinophthalmoscope," from κίνησις (motion). It has served us in our investigations concerning the physiology of the movements of the eyes (*Arch. d'Opht.*, 1891, p. 385) by showing that, even in a normal condition, the eyes move by jumps and not continuously.

## TREATMENT OF INSUFFICIENCY OF CONVERGENCE AND OF DIVERGENT STRABISMUS.

Insufficiency of convergence and divergent strabismus are so intimately related to each other, and the lines of proceeding in the two conditions are so similar, that we may consistently combine in the same article the treatment of both affections.

It is self-evident that this treatment will be, first of all, *etiological.* It is true that when the divergence is due to a *cerebral lesion* our therapeutic resources are often very limited.

In *tabes* the paralysis of convergence, as well as that of the ocular muscles, is sometimes seen to disappear spontaneously.

*Hysteria* and *neurasthenia* are more accessible to medical intervention. This is not the place to discuss in detail the multiple means which may be employed in these complex affections. The essentials will always be *physical, intellectual,* and *moral hygiene,* a rational and comforting regimen, out-of-door exercise, good nourishment, and the like. Hydrotherapy, judiciously applied, almost always does patients of this kind much good. It is

[1] Landolt, Soc. française d'Opht., 1892, p. 258.

often advisable to remove them from their ordinary environment, to make them travel, or, at least, change their residence. Other surroundings give rise to other ideas; from calm mountain heights, the annoyances, cares, and ambitions of a busy life appear small or disappear altogether, like the human habitations one has left behind, and which are scarcely perceptible below one's feet. Considered from the other side of the ocean, a question may change its aspect, and often shows us its better side.

These considerations are of special importance in the case of asthenopes whose malady is due to overwork. It is for them especially, that repose of the mind and of the eyes, as well as change of habitat, hydrotherapy, etc., have the best influence.

Evidently these good influences can do no harm to those whose insufficiency of convergence is due to a *lack of use* of their adductor muscles, though they will not effect a cure. It is the *exercise* of the impotent function more than anything else which will render them service.

In this connection it seems appropriate to attempt an explanation of the difference of opinion among equally competent authors. Some of them, as Noyes, for instance, recommend these exercises in insufficiency of convergence and incipient divergent strabismus.[1] Others, like von Graefe, advise the opposite, saying that the efforts required of the adductor muscles would only fatigue them and weaken them still more.

To us it seems that in different conditions each opinion may be correct. Thus, von Graefe is certainly right for many cases of our first and second forms of insufficiency. On the other hand, proper exercises may be indicated in cases of the third form, where the infirmity is ascribable to a lack of exercise, and, moreover, in cases where, after surgical intervention, the adductor muscles are prepared to resume their function.

When there is only insufficiency, these exercises may be performed with a clear, small object, a black spot on a white background, or, better still, with the luminous aperture of our dynamometer. This object of fixation is gradually brought nearer the patient's eyes along the median line, or, what amounts to the same thing, the patient approaches the object, taking care to overcome as long as possible the tendency to double vision, which becomes increasingly manifest as the test-object approaches the *punctum proximum* of convergence. It is precisely for the sake of rendering this diplopia clearer, and, on the other hand, to aid fusion, that it is best to choose as a fixation-object, not a finger or a pencil, but a very fine luminous point or luminous line.

In the case of *commencing divergent strabismus*, exercise of this sort may still be of service, but only on one condition,—that is, that binocular vision exists. Hence high degrees of strabismus and cases in which one of the eyes is markedly amblyopic or amaurotic are from the outset excluded.

If binocular vision is lacking, but the sight of both eyes is fairly good,

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[1] Compare, likewise, R. Cross, Asthenopia, Bristol Med.-Chir. Journal, p. 181, 1893.

the treatment should begin by *stereoscopic exercises* for the purpose of re-establishing fusion of the retinal images. The stereoscopic objects, at first widely separated from each other, are gradually brought nearer, with the view of bringing the eyes towards parallelism and thence to increasing convergence. We follow here the converse principle from that which guided us in the treatment of convergent strabismus. In that case, we took care to exclude, by means of the strongest convex glasses, any effort of accommodation, for the sake of diminishing at the same time the impulse to convergence. Here, on the contrary, we give the patient the weakest convex glasses with which he can still distinguish clearly the stereoscopic objects, and we diminish their strength in proportion as, by the diminution of the distance between the objects, the convergence increases.

There may even present itself a case of so high a degree of myopia that the patient does not need glasses, or even needs concave glasses, in order to see distinctly to the bottom of our stereoscope. In such a case, an increase in the power of the concave glasses will stimulate his convergence.

I beg permission to cite here an observation well adapted to demonstrate the utility of such exercises. I noticed one day in the waiting-room of my clinic a woman whose divergent strabismus was striking at the first glance. I had no doubt that it was with reference to this infirmity, that she had come, but she declared that her visit was only to bring her child, suffering from an ophthalmia; that, as for herself, she had squinted too long to think of seeking treatment for it, and that she did not desire it. However, I got permission to examine her.

I found, in the left eye, myopia 0.25 D., $V = 1$, and in the right eye, myopia 0.5 D., $V = 0.9$. The latter eye diverged through an angle of thirty-nine degrees; the fields of fixation of the two eyes did not exceed thirty-five degrees to the nasal side; the temporal side was also of less extent than in the normal condition.

On making the patient look at a distant candle-flame, she did not see double, either with or without a colored glass, but crossed diplopia appeared when a vertical prism was placed before one of her eyes. Hence there was suppression of the impression received by one of the retinæ in the horizontal meridian, the parts above and below having maintained their relations with the retina of the other eye.

The patient being emphatically requested to fix a near object, she made a few efforts of convergence, but the eyes were far from acquiring the proper direction, and the right eye again resumed at the end of an instant its divergence. Nevertheless, we encouraged the patient to undertake stereoscopic exercises.

She acted on this advice, but without much enthusiasm. It was rather with a view to please us, that she looked into the stereoscope, while present for the sake of her child's treatment. In spite of that, we succeeded in re-establishing binocular vision and in achieving a complete disappearance of the strabismus at the end of two months. The normal condition persisted for several months,—that is to say, so long as the patient continued occasionally the stereoscopic training. She negligently abandoned it, not caring, as she had declared at the outset, to be cured. But, for our part, there exists no doubt that this person would have achieved a perfect cure of her strabismus if she had continued the stereoscopic training regularly, and had substituted for it later direct exercise of the convergence.

However this may be, the result proves that, even in the case of a divergent strabismus of long standing and of high degree, orthoptic training may have a very favorable influence. There is all the more reason why such training should exert a beneficial influence when strabismus is beginning, and especially in insufficiency when due to a lack of use.

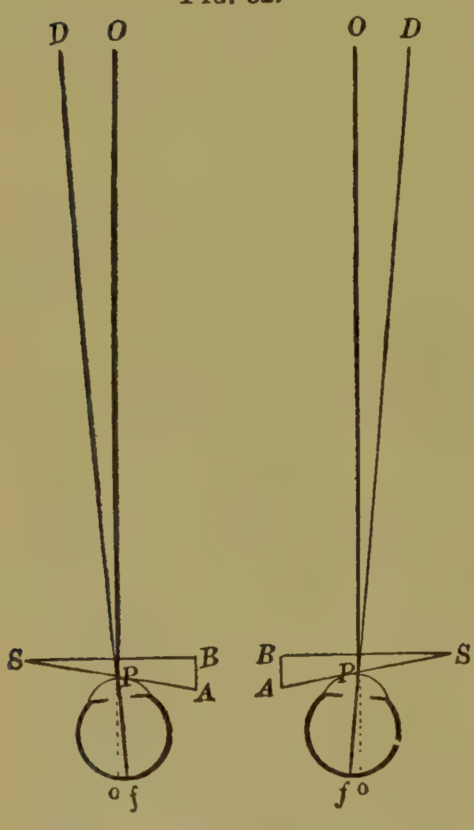
Such training may also be undertaken in certain cases of *muscular insufficiency*. But in most instances it is better to cause the orthoptic exercises in such circumstances to be preceded by a more radical and powerful remedy, that is to say, by operation, of which we shall speak later.

All these treatments, general and orthoptic, even under the most favorable circumstances, demand much time. Hence it has been sought to solace the victim of asthenopia or insufficiency of convergence by means of *palliatives* until his infirmity shall have disappeared. In short, it has been sought to *diminish the convergence* required for his work.

Evidently, the simplest means of achieving this is to *increase the distance between the eyes and the fixation-object.* This is so easily done, indeed, that most asthenopes are found to have naturally adopted it even before they consult us. It is only myopes, whose *punctum remotum* is situated very near their eyes, that we can sometimes relieve with concave glasses, which, by increasing the extent of their vision, permit them to read and write with less convergence.

Thus, the myope of 4 D. is obliged to make an effort of convergence of $4ma$ in order to work at the distance of his *punctum remotum.* And, as the experiments above mentioned have taught us, he needs twice as much as this,—that is to say, $8ma$, in reserve, or a total of $12ma$ of convergence power. By giving him concave glasses of 1 D., we carry his distinct vision to $\frac{1m.}{3}$ Therefore we have diminished by one metre-angle the convergence necessary for fixation, and by $3ma$ the total required for continued labor.

FIG. 52.




But practice proves that the applicability of concave glasses for this purpose is very limited. If the degree of the myopia be high, the glasses necessary to lessen effectively the convergence are so strong that the reduction which they produce in the size of the retinal images becomes a new cause of fatigue to the patient.

Hence attempts have been made to diminish the convergence by other optical appliances,—that is to say, by *prismatic glasses.*

If we place in front of one eye or in front of each of them a prism whose apex is directed towards the temple, as is shown in Fig. 52, rays coming from the distant object $O$, instead of continuing their course in a straight line towards $o$, will deviate at $P$, towards the base of the prism, in the direction $Pf$. The eyes, instead of being directed in a parallel direc-

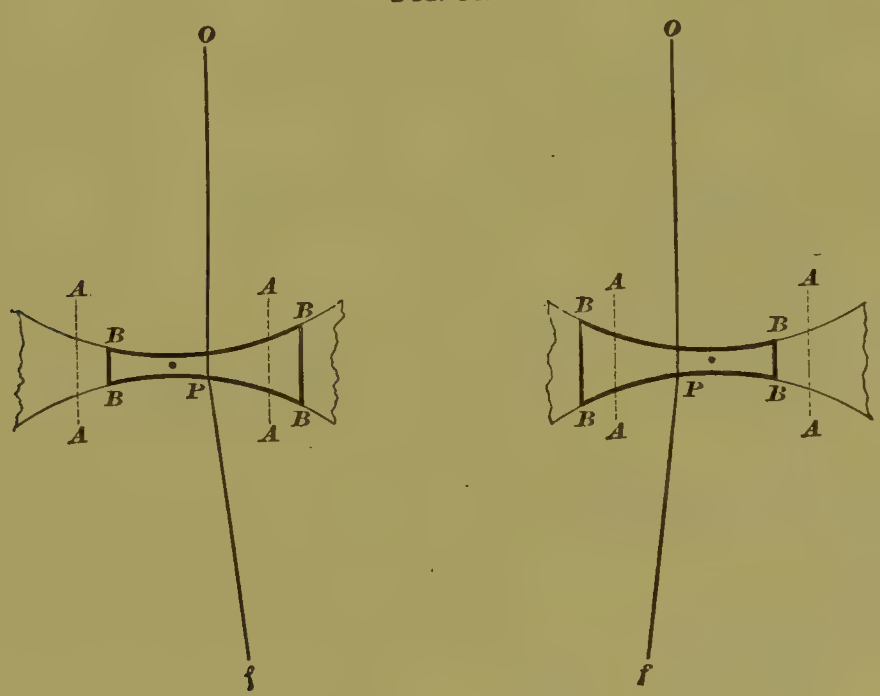
tion towards $O$, during distant vision, will be directed towards $D$, that is to say, they will diverge through an angle equal to twice the angle $DPO$. It is for this reason that the name *abducting* is given to the prisms placed with the apex towards the temple.

If the object be brought nearer, the convergence required for its fixation will always be less by this angle than it would if the individual were not using these glasses.[1]

It is evident that, if the patient also require concave or convex spherical or cylindrical lenses, the two surfaces of the prism may be utilized for this purpose. Thus it becomes possible to combine the two optical appliances before mentioned for the diminution of convergence,—concave lenses to increase the distance of the *punctum remotum*, and prisms to modify the direction of the visual lines.

For the same purpose, spherical lenses may be *decentred;* that is to say, they may be placed before the eyes in such a way as to use an eccentric part, which has the same effect as the prism combined with a spherical glass.

FIG. 53.



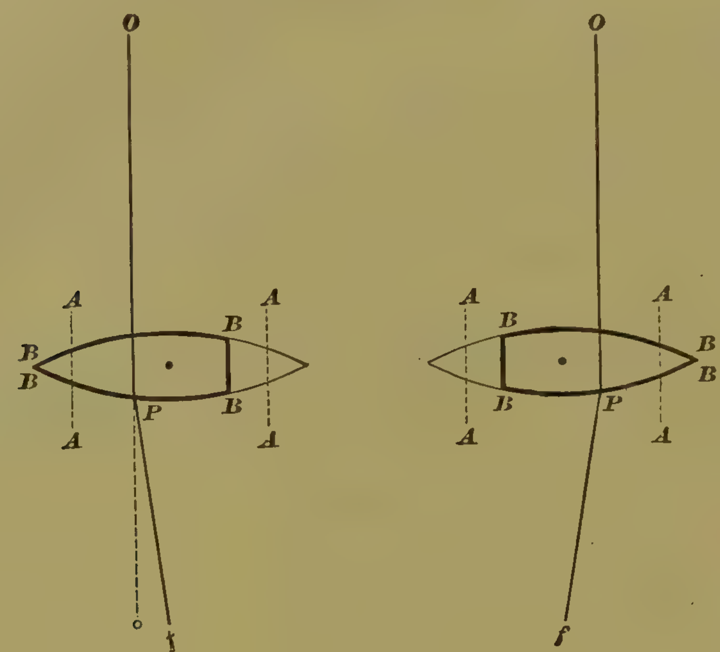

Thus, in Fig. 53, instead of cutting the concave spectacle glass with its centre coincident with the optical centre of the lens,—that is to say, along $AAAA$,—it may be cut from an eccentric portion, along $BBBB$. The luminous rays coming from $O$ are then deviated at $P$ towards the base of the prisms thus obtained,—that is to say, towards $f$. In this way, the eyes that use these glasses are obliged to diverge ($fP$) when looking at a great distance ($PO$), or to converge less when looking at near objects.

To obtain the same effect with convex glasses, the patient must be made,

[1] Consult in this connection the treatise of Maddox, The Clinical Use of Prisms, Bristol and London, 1893, and Prentice, vol. ii. of the present work.

on the contrary, to look through the outer part; the glass should be cut, as is shown in Fig. 54, so that the centre of the glass when mounted in a frame will be farther out than is its optical centre (*BBBB* instead of *AAAA*). Here, again, the convergence is diminished for each eye to the extent of the angle *oPf*.

Fig. 54.




Great hopes were entertained as to the efficacy of prisms in the treatment of motor asthenopia. These have not been entirely realized, for reasons which our calculations explain.

Indeed, we have seen that a metre-angle corresponds to a prism of 1° 40′ (No. $3\frac{1}{2}$, according to the old numeration) placed before each eye. This is about the limit of the prism that can be used in practice. Otherwise the glasses are not only too heavy, but the chromatic aberration and the deformity of objects which they produce become too troublesome.

The aid that can be given to a patient by means of prismatic glasses is therefore scarcely a metre-angle and a half. The effect of decentring lenses is often still less. Now, the insufficiency which gives rise to asthenopia is generally due to a defect of the convergence power much greater than one or two metre-angles.[1]

We do not deny the utility of prismatic glasses, or of decentring in low grades of insufficiency, but in high degrees they have no value. In the former case it is even essential to guard one's self from judging by appearances, as these cases often get well of themselves.

I may quote here an observation well fit to instruct us concerning the efficacy of prisms in asthenopia. On examining one day the spectacles of a patient who had come to consult me for something different from asthenopia, I found that she had been wearing simple prisms with their *apices towards the nose*. These spectacles, she told me, had been

[1] Landolt, Insufficiency of the Power of Convergence, Ophth. Rev., p. 190, 1886.

prescribed for her many years before by Dr. L., and they were absolutely indispensable for work.

At my request the patient found the formula for her glasses, in which prisms were prescribed, but their apices were to have been in the opposite direction,—that is to say, *towards the temples*. The master, for he was one of the most competent of ophthalmologists, who had prescribed the glasses had therefore hoped in this way to correct an *insufficiency of the internal recti*, as it was called in those days. But the optician had made a mistake, had mounted them in the opposite direction, and the patient had worn them and found them so satisfactory that she affirmed her inability to work in comfort without them. What is the conclusion to be drawn from this tale? Is it that, the doctor having mistaken an insufficiency of divergence for an insufficiency of convergence, the patient's guardian angel had so controlled the hands of the optician that the latter, by making a counterbalancing mistake, had set things in order? For my part, I see no other conclusion than the slight effect of weak prisms and the great power of imagination.

If rest and general treatment are shown to be ineffective in insufficiency of convergence, and if it be of too high a grade to be susceptible of correction by optical means, one is justified in having recourse to surgical intervention in order to re-establish the lost power.

From the days of von Graefe's to our own, *tenotomy* has been the usual operative procedure for remedying asthenopia. In insufficiency of convergence, the external recti are divided, for the purpose of diminishing the work of the adductor muscles by weakening their antagonists.

The harm that has been done by these tenotomies is considerable. Not only has asthenopia very rarely been cured in this way, but a most troublesome convergent strabismus, with homonymous diplopia, has often been added to the former trouble.

In the surgical treatment of insufficiency of convergence, two operations should be taken into consideration: tenotomy of the abductors and advancement of the adductors.

The former operation diminishes the power of divergence, the latter increases the power of convergence. The first of these two operations is the simpler. But to know whether it is applicable, we must first ascertain if the patient possesses divergence strong enough to allow a part to be sacrificed in the interest of convergence. If in such a case as is represented by the letters *F* and *G* of our diagram (Fig. 48) we diminish by a tenotomy the already defective divergence, the minimum of convergence is rendered positive; in other words, a convergent strabismus is produced.

Hence it follows that tenotomy is out of the question in cases wherein divergence is zero or less than the normal. This is, as has been said before, very common.

If the divergence be normal, as in *D* (Fig. 48), or exaggerated, as in *B*, *C*, *I*, *K*, should tenotomy always be done?

That depends upon what can be accomplished by it. If diminution of the divergence without the sacrifice of all this function suffices to supply the deficit of convergence, then tenotomy of one or both of the abductors may be indicated. But if it is not sufficient, then this operation is at all events useless: asthenopia would persist in spite of it.

Experience only can solve the problem of the efficacy of tenotomy. It has in our hands given the following results: When the conditions of the motility of the eyes are otherwise good, the excursions about normal, the amplitude of convergence only slightly limited, being, as it were, displaced towards the negative, as in $B$ and $C$ (Fig. 48), then tenotomy gives good results; that is to say, a partial limitation of divergence may increase convergence to the required amount.

Let there be, however, no mistake: The tenotomy of which I am speaking, and which I consider as admissible in certain cases of insufficiency of convergence, although involving the entire extent of the muscle's insertion, ought to be a simple *tenotomy*, limited to the strict detachment of the tendon from the globe, but *not a setting back of* the muscle, as is obtained by disengaging the muscle from the surrounding tissues. In the latter way one markedly diminishes the excursion of the eye towards the side of the operated muscle.

This fact is capable of the following explanation. Tenotomy thus practised does not act so much by the displacement of the insertion of the muscle as by the weakening of its action. Indeed, the new attachment of the muscle to the ocular globe is usually not so firm as its original insertion. It takes place by the intermediation of a more or less lax cicatricial tissue, which holds the muscle in place relatively to its surroundings, but sensibly changes its influence upon the eyeball.

Let us cite here a case of motor asthenopia, in which tenotomy had a very favorable influence.

FIG. 55.


The *left* vertical line of Fig. 55 represents the amplitude of convergence *before* the operation. It is to be analyzed as follows:

$$\left.\begin{array}{l} p = 7 \\ r = -3 \end{array}\right\} a = 10ma.$$

Tenotomy of one of the external recti, followed by exercises in convergence, brought nearer, so to speak, the whole of the amplitude of convergence. It became: $\left.\begin{array}{l} p = 10 \\ r = 0 \end{array}\right\} a = 10ma$ (*right* vertical line, Fig. 55), and the asthenopia was cured.

In other still more favorable cases the total of the amplitude of convergence not only remains intact after a proper tenotomy, but may even gain somewhat when it is followed by methodical training.

But it would be wrong always to expect such happy results from tenotomy. Although at times the amplitude of convergence remains intact after this operation and becomes more favorable for near work, and even in some cases may increase, yet diminution of amplitude is, as a rule, the consequence of the tenotomy.

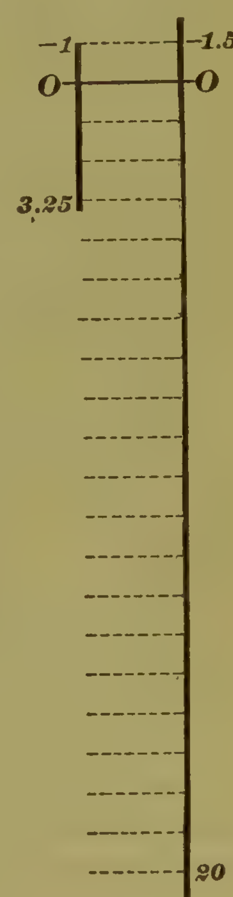
This happens when a notable reduction of this range—even the limitation of the fields of excursion—shows an insufficient development or great weakness of the motor system of the eyes. In this case the sacrifice of a

great part—even of the whole—of the divergence increases the positive convergence only very slightly. The amplitude of convergence remains reduced, and to the insufficiency of convergence which persists, is added an insufficiency of divergence, or, more correctly speaking, convergent strabismus with most troublesome homonymous diplopia. The patient sees double during either near or distant vision; the operation which was to have cured him has greatly aggravated his condition.

It is to escape this danger that we generally abstain from performing tenotomy for the cure of motor asthenopia. Loyal to our principle of *increasing the power of the weak muscles rather than decreasing that of their antagonists*, we have for many years had recourse in such cases to the advancement of the *internal rectus* of one of the eyes. Double advancement is only exceptionally necessary. We have found, indeed, that *by muscular advancement much more is gained on the positive side of the amplitude of convergence than is gained by tenotomy, while nothing is lost on the negative side.*

Let us cite in support of this the following observation:

FIG. 56.




A boy ten years old, slightly myopic and astigmatic to 0.75 D., but having excellent visual acuity in both eyes.

Asthenopia which has lasted a long time, in spite of glasses, general and local care, which the best physicians and specialists of this country have given him in profusion.

His amplitude of convergence analyzes as follows:

$\left.\begin{array}{l} p = 3.25 \\ r = -1 \end{array}\right\} a = 4.25ma.$ It is shown in *left* vertical line of Fig. 56.

Energetic advancement of one of the internal recti.

Three weeks later the amplitude of convergence was found to be $\begin{array}{l} p = \text{more than } 20ma \\ r = -1.5ma \end{array}$. In other words, the advancement procured for him a converging power much stronger even than the normal, and left his divergence intact (*right* vertical line, Fig. 56).

This result has remained permanent. The young man has finished his studies brilliantly, and when I saw him thirteen years later, his amplitude of convergence was still as extensive.

We could cite a number of quite as conclusive observations as this, proving the superiority of advancement over tenotomy in motor asthenopia.

It is self-evident that in deciding to intervene surgically in a case of insufficiency of convergence we do not renounce the other curative means which have already been discussed. We always consider them as indispensable auxiliaries with which to achieve a complete and lasting cure.

## TREATMENT OF DIVERGENT STRABISMUS.

The principles which we follow in the treatment of divergent strabismus are the same as those that have guided us in that of insufficiency of con-

vergence. We shall only apply them with more energy. Thus, one will not hesitate long about intervening surgically. The best operation here also is *muscular advancement*, and it should generally be performed on both adductors.

When the strabismus is of high degree and of long standing, especially when it occurs in an amblyopic eye, it will be best to practise at one sitting the advancement of both internal recti, combined with the resection of their tendinous extremities. If after weeks or months the effect of this operation should still be insufficient, the moderate tenotomy of one external rectus, or even of both external recti, may be added. But this is necessary only in extreme cases of divergent strabismus.

After every muscular advancement we always bandage *both* eyes and keep them thus for at least five days, in order by their immobility to favor the attachment of the muscle.

In convergent strabismus this suppression of vision by bandaging is very useful, for it diminishes the tendency to convergence, which might thwart the effect of the operation. In divergent strabismus the efforts of convergence are, on the contrary, favorable, inasmuch as they tend to the same end as does the operation,—that is to say, to the diminution of divergence. For this reason we leave off the binocular bandage in this case sooner than after an operation for convergent strabismus. After the removal of the bandage orthoptic exercises are immediately instituted. They tend, at first, to re-establish binocular fusion by means of stereoscopic exercises. Then the patient is taught to bring a small luminous object slowly towards his eyes, just as in the orthoptic treatment of insufficiency of convergence.

If the operation should have accomplished too much, which happens very rarely and only when the advancement of the internus has been combined with tenotomy of the externus, the set-back muscle must be advanced by means of one or two sutures.

Unfortunately, one often sees operators even yet try to correct divergent strabismus by tenotomies alone. They are, generally, obliged not only to do what I have called exaggerated setting back of the muscle,—that is to say, to detach the muscle from its connections with surrounding tissues,—but also to repeat this operation several times. Many of them have recourse to what Graefe called a *thread-operation*, which consists in drawing the eye forcibly by means of a suture towards the nose, with a view to obtaining an insertion of the external rectus still farther back. We again repeat what we published twenty years ago and what we have just said in discussing the operation for convergent strabismus, that this procedure is unworthy of modern ocular surgery. If one succeeds in thus giving to the eyes a parallel direction when they are directed straight forward, one creates an insufficiency of the abductors, a convergent strabismus with homonymous diplopia when binocular vision exists, in the lateral excursions. The eye has been partially removed from its muscular funnel instead of

being put into it, as is done by advancement, or instead of being turned in it, as by advancement combined with tenotomy.

I have published in the *Archives d'Ophtalmologie* (March and December, 1895, and July, 1896) a series of characteristic observations concerning the various methods of operation in the different forms of strabismus. May I be allowed to end this paragraph with a case that occurred to me while I was correcting the proofs of this work? It concerns a man twenty-two years of age. His *left* eye showed: H. 0.5; V = 1; the *right* eye, M. 2.5 with AS. m. 1.5; V = 0.3.

From the first years of life he was affected with a *divergent strabismus* of his *right* eye, amounting to 26°.

The *temporal* excursions of both eyes were very extended (65°), the *nasal* ones notably limited (43° on the left, 40° on the right eye).

There was neither simultaneous vision nor diplopia, nor, of course, macular fusion.

I performed at once the *advancement of both internal recti muscles* with the resection of their tendinous extremities.

Six days after, the sutures were removed. The seventh day, visual exercises were begun with a distant candle-flame, the left eye being provided with a colored glass, the right with its correcting lens.

After a short time the patient manifested a crossed diplopia of 2°. With a prism of 2° he saw the two images at the same place, but without fusing them as yet. The same thing happened with the stereoscope: the two engravings were seen at first only simultaneously, but after some exercises the patient fused them in such a way that he perceived distinctly the perspective,—*i.e.*, the *relief*.

In Hering's test with the falling balls, all his answers were correct.

Continuing regularly the stereoscopic exercises, the patient recovered thus a perfect binocular vision and, with it, the ideal cure of his strabismus.

Three weeks after the operation the maximum of convergence ($p$) was more than 20*ma*, the minimum —1.5*ma*, therefore the amplitude of convergence was 21.5*ma*.

This fact is extremely significant. It shows that the advancement of the abducting muscles has given the patient a converging power which considerably surpasses the normal one, and that *without damage to the abduction*, which is equally superior to the normal.

In the same way, far from losing, as after setting back, somewhat of the associated lateral movements, he has gained by the advancement of the muscles. The nasal excursions are now equal to 50° (instead of 43° and 40°), whilst the temporal ones pass still beyond 60°, as before.

## NON-PARALYTIC STRABISMUS SURSUM AND DEORSUM VERGENS.

Leaving out of consideration the elevation of an eye which is strongly deviated inward, and the lowering which often accompanies high degrees of divergence, manifest vertical strabismus is very rare. Generally, strabismus sursum or deorsum vergens is due to the paresis or paralysis of a depressor or levator muscle, or to congenital absence of the muscle (Lawford). A badly performed strabotomy may likewise give rise to a difference in level between the eyes.

But vertical deviations may exist in a *latent* condition.

Dr. George T. Stevens has especially drawn attention to this form of motor troubles of the eyes and to their consequences. We would refer our readers to the chapter of this work in which that author himself has given the results of his investigations. We here limit ourselves to saying that latent vertical strabismus shows itself especially by asthenopia. It is de-

tected by the procedures which serve to make known the so-called "direction of repose" or "equilibrium" of the eyes.

In order to measure the degree, the examiner provokes, by means of strong abducting prisms, a homonymous diplopia which the patient is incapable of overcoming. Binocular vision being thus rendered impossible, the eyes promptly abandon themselves to the difference in height, the natural correction of which has caused so much pain. The vertical diplopia serves to determine the degree of the deviation in height, according to the methods which have been described above. That is to say, the prism which, placed vertically before one of the eyes, brings the two images to the same level measures the degree of the strabismus.

Let it be said, parenthetically, that one readily sees here how much more rational it is to express the action of the prism by the angle of the deviation which it produces than by metre-angles. The *metre-angle*, which measures the degree of convergence or divergence between two eyes separated by a base-line, has no *raison d'être* when it is a question of elevation or lowering, in which case the two eyes may be considered as united by their centres of rotation.

It is, of course, indispensable that the edges of the abducting prisms should be exactly vertical, and the edge of the measuring prism, as well as the line joining the centres of rotation of the eyes, exactly horizontal; otherwise one incurs the risk of provoking a vertical diplopia in healthy subjects, or of correcting it in cases where it exists.

Vertical strabismus never attains as high a degree as does convergent or divergent strabismus. Hence one often succeeds in correcting it by means of vertical prisms. It is well to divide the prismatic effect between the two eyes, turning the two apices in opposite directions.

If the defect require a stronger prism than one of three degrees, *operation* is generally necessary in order to correct it. If the ocular excursions are good and are equal in both eyes, the *tenotomy of the superior rectus of the higher eye* often suffices to remedy the asthenopia created by the strabismus.

But it is nowhere more important than here to take into account the counsel of prudence which has been above formulated with reference to tenotomy. A slightly felt setting back of the superior or inferior rectus muscle, even though perfectly correcting the strabismus for a certain direction of vision, may easily bring about another strabismus so soon as the eyes move towards the sphere of action of the operated muscle. It is for this reason that, if a tenotomy appear admissible, we prefer to perform it on the levator rather than on the depressor. This is because, the eyes being more often used in looking *downward*, insufficiency of a levator is less troublesome than is that of a depressor.

If the difference in height is very marked, and especially when examination of the excursions demonstrates a gap in the lower part of the field of fixation of one of the eyes, we perform the *advancement of the inferior rectus* of this eye, according to the principles already explained.

Vertical strabismus is generally much more rebellious to treatment than horizontal, and in the higher degrees one may be obliged to operate on both eyes in order to remedy a strabismus sursum or deorsum vergens.

Our method of procedure for correcting a difference in height between the eyes may be summed up as follows:

The eyes being generally employed in looking downward, it is better to lower the more elevated eye than to elevate the lower one. This lowering may be obtained in small degrees by tenotomy of the superior, but it is usually preferable to advance the inferior rectus. In the highest degrees the combination of advancement with tenotomy may become necessary.

It will be noticed that I operate only on the recti muscles. In fact, though I was the first to perform the *section of an oblique muscle*,—that of the inferior at its origin,[1]—I am far from advising this operation with a view to remedy, for instance, paresis of the inferior rectus of the other eye, notwithstanding the fact that this oblique is the antagonist of that rectus. We perform the section of the inferior oblique only in the very rare case in which we desire to bring to the middle of the palpebral opening the upper part of the cornea, when it alone has remained transparent and has had an artificial pupil made opposite it. The combination of all the means at our disposal—tenotomy of both levators and advancement of the inferior rectus—may then be necessary in order to obtain as considerable a lowering of the eye as is requisite.

It is unnecessary to add that here, as in all strabotomy, careful watching of the patient, binocular bandaging, etc., are essential, and that orthoptic training will aid us in controlling and confirming the effect of our operation.

### NYSTAGMUS.[2]

Nystagmus consists of an *oscillatory, involuntary, rhythmic movement of the eyeball, having small range, and being more or less continuous.* This movement may take place in the horizontal direction (*horizontal* nystagmus), in the vertical (*vertical* nystagmus), or may represent a more or less extended rotation around the line of sight (*rotary* nystagmus). Sometimes it affects the direction intermediate between the horizontal and the vertical (*oblique* nystagmus). Finally, the oscillations of the globe, while remaining rhythmic, may simultaneously represent different types of ocular movements, in which case the nystagmus is called *mixed.*

Nystagmus is generally binocular and associated, at times it is unilateral. In the latter case it is almost always vertical.

In nystagmus, properly so called, the rhythmic oscillations are produced in all directions of vision. Their range and rapidity, however, vary under the influence of certain causes. They stop during sleep; they are more pronounced when the victim of them is aware that he is being

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[1] Landolt, La ténotomie de l'oblique inférieur, Arch. d'Opht., 1885, p. 402.

[2] See Landolt and Eperon, loc. cit., p. 932.

watched than when this is not the case, and are also more marked during fixation than when the gaze is inattentive. Nystagmus may increase or diminish during a certain direction of the gaze or in certain positions of the head; it is at times entirely suppressed by an unusual nearness of the object of fixation. In this case it is the convergence of the eyes that produces this effect; in fact, adducting prisms exercise the same action.

The oscillations of the ocular globes are often accompanied by analogous movements of the head. These latter, as well as the movements of the eyes, are made round axes which are parallel to each other, but the rotations of the head are in the opposite direction to those of the eyes. (Alf. Graefe.)

Apart from this anomaly, the rotations of the eyes may be executed with regularity in all directions, and the binocular movements are notably free from disturbance when no strabismus exists. However, the field of fixation, whether monocular or binocular, is notably reduced, especially in certain directions.

From the point of view of etiology, and also of symptomatology, a distinction must be made between congenital and acquired nystagmus.

The former develops either very soon after birth or in early infancy. It is almost always associated with a *weakness of visual acuity.*

The frequent relation between albinism and nystagmus has long been known, and it is to be readily explained by the imperfection of vision in eyes devoid of chorioidal pigment. Nystagmus may, however, be coincident in certain cases with good visual acuity. It then manifests itself only when the vision is inattentive, and disappears during fixation. With certain subjects an hereditary influence has been determined. (Alf. Graefe, Lloyd Owen.)

Persons affected with congenital nystagmus, when they are in a condition to give satisfactory accounts of their vision, do not complain of any trouble of the latter which is specially imputable to the infirmity in question. It is noteworthy that they perceive no apparent motion of objects, and are annoyed only by the weakness of their visual acuity. In exceptional cases, where the acuity is good, binocular vision exists, the limits of the field of fixation are normal, and the associated excursions of the eyes, as well as the amplitude of convergence, leave nothing to be desired.

It is quite otherwise in *acquired* nystagmus, which we ought to subdivide into two categories, *essential, idiopathic* nystagmus, and the nystagmus which is *symptomatic of lesions of the central nervous system.*

The former—essential nystagmus—is, so to speak, a malady of occupation; it affects especially miners, and of them principally those who work in coal-mines. Attention having been called to it as long ago as 1861 by Decondé, miners' nystagmus has been studied especially by Schroeter, Mooren, Nieden, Snell, Dransart, and Romiée.

Since nystagmus is much too frequent in the large population engaged in the mining industry, we have asked our eminent colleague and friend

Simeon Snell, F.R.C.S., of Sheffield, to communicate to us the results of the investigations which he has made on this subject in the mines of Great Britain. The following, somewhat abridged for this article, is the interesting reply which he has kindly furnished:

"Nystagmus, as occurring in miners, is characterized by the apparent movement of objects, either in a circle or ellipse; headache is often present, and especially giddiness, which sometimes causes the miner so to stumble about that he is compelled to leave his work in the mine. The movements of the globe are chiefly rotatory, and, though to-and-fro oscillations are sometimes superadded and are very rarely vertical, the first named are seldom if ever absent. The rapidity of the ocular motions varies greatly: from sixty to one hundred and fifty motions may be counted in a minute; I have observed them as frequent as three hundred and fifty. Both eyes are affected; the rapidity of movements may vary in the two eyes. The more rapid the oscillations, the less extended is the excursion of the globes. The oscillations are arrested by turning the gaze downward below the horizontal line. Miners often rest their eyes in this way. Looking upward (levator muscles of the globe), and especially obliquely to one side or to the other, rapid movements of the head, lowering of the head and suddenly raising it, are means of increasing the rate of movements of the eyeball, or, in other cases, of rendering them evident. Placing the patient in the position he would assume at his work is another method. Associated frequently are tremors of the head (noticeable to the hand placed on the head), of the eyelids, and of the muscles of the face and neck; torticollis is met with; night-blindness has been alleged to be present. Nieden and Romiée and myself dispute its presence: the nystagmus alone is a sufficient cause for any difficulty in seeing in a failing light. Errors of refraction, myopia, hypermetropia, and astigmatism are often present, but bear no causal relation to nystagmus; visual acuity is generally unaffected. Color-perception is good, and, so far as the oscillations permit of testing, the field of vision is normal. The onset of the disorder is often brought about by some attack of illness. It is generally met with in men who have worked in the mine for some years. Ninety per cent. of cases occur in persons from twenty-five to forty-five years of age.

"Nystagmus is found in miners (coal-getters) engaged at the coal-face and who work in a more or less constrained position of body and of eyes. It is desirable to get coal in as large pieces as possible, and to do this they undercut, or 'hole' the seam.[1] A man sits with his legs crooked up lying almost on his side and strikes the coal with a horizontal swing of his pick at the bottom of the coal-seam. He will cleave away the coal to a height of from eighteen inches to two feet, and then as he gets deeper in he draws his body under the coal, lying on one side or the other. The distance he may undercut the coal varies considerably. The process is called 'holing,'

[1] Miners' Nystagmus, by Simeon Snell, F.R.C.S., Wright, Bristol.

and sometimes the undermining may be continued from two or three feet to as much as seven or eight feet. The miner applies timber supports to keep the coal from falling as he proceeds with his work. As just described, it is called 'bottom holing,' but the seam may be attacked in the middle (middle holing) or at the top (top holing). A miner engaged at this work will direct his gaze to different parts as it becomes necessary for him to strike, for the eyes will follow the pick point, but the tendency will be for the gaze to be directed upwards (using the ocular elevators) more or less obliquely. He will lie sometimes on one side and sometimes on the other; his legs will be crooked up, his head thrown back and flexed more or less on the shoulder beneath. This position is shown in the photographs (Figs. 57 and 58), which were taken in the mine with a magnesium flash light, of a man whilst actually at work. Ninety-eight per cent. of all cases of nystagmus coming under my observation have been occupied at the coal-face and more or less engaged in this kind of work. The thickness of the coal-seam varies greatly in different parts, but work of a very similar kind is done in coal-mines in all countries.

"Besides the coal-getters, there are others in a mine who attend to the roads, fill the wagons, push these 'trammers,' or drive the ponies. There are also 'deputies' or 'overlookers,' whose work it is to see to the safety of the places the men work in, both as to freedom from gas and as to the condition of the roof of the mine. These latter occasionally suffer from nystagmus, and if the work be analyzed it will be found to necessitate the same oblique, upward direction of the gaze, and generally these men have previously worked as coal-getters. The photograph (Fig. 59) shows a 'deputy' examining the roof to ascertain its soundness by striking it with his stick. It must be remembered that the height of the working-places and passages in the mine is nearly always so low that this alone compels a constrained attitude. 'Onsetters,' whose duty it is to see to the ascent of the full and descent of the empty coal-tubs, sometimes get nystagmus, as do also 'timbermen.' It may be accepted as a rule that all cases of nystagmus occur in those who are either working as coal-getters or who have done so, or that the work in which they have been employed has been one necessitating an upward look of the eyes for more or less prolonged periods.

"What has been said will point out the direction in which the etiology of the affection must be sought. Nystagmus depends on a chronic weariness induced in the levator muscles of the eyes by the constrained position. Like effects are found in other muscles of the miner, producing torticollis, tremors of the head, and quivering of the eyelids. It is thus similar to other occupation neuroses, and in the same category as those met with in writers, compositors, telegraphers, ballet-dancers, and many others.

"Nystagmus, besides possessing the peculiarities already mentioned, occurs with all kinds of lighting. I have met with it in workers with safety-lamps, candles, large open lamps, and when the artificial light was good. On the other hand, there is some reason for believing that the strain is

FIG. 57.

FIG. 58.

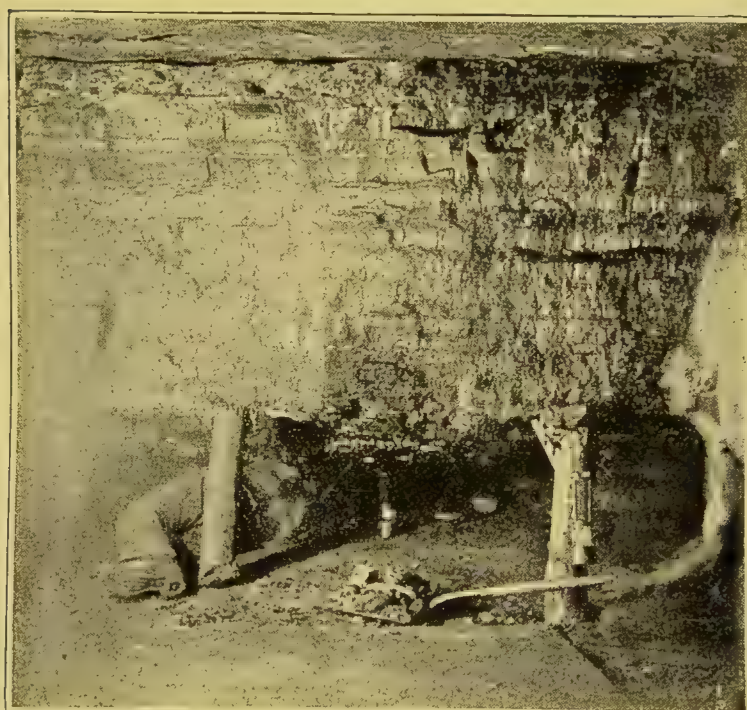
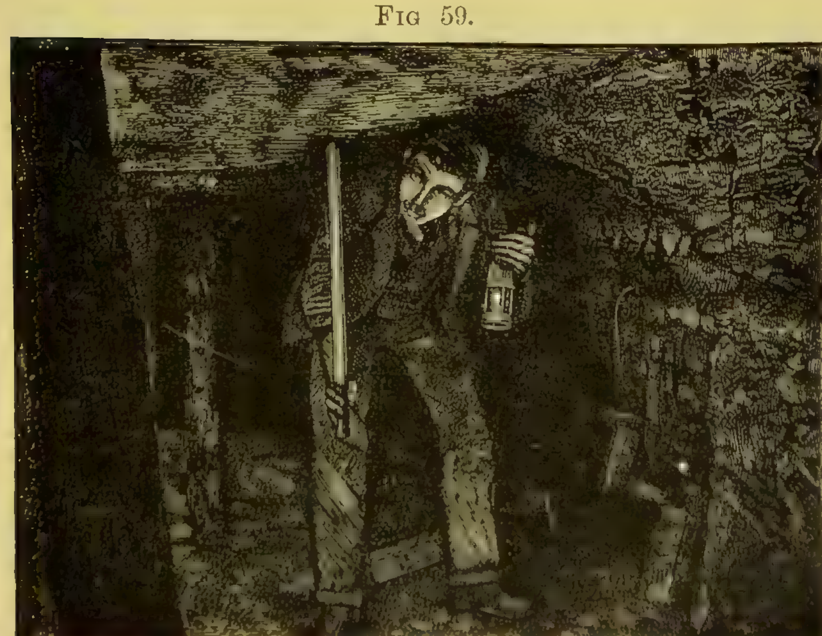


FIG 59.



Figures illustrating positions of miners while at work.

greater the worse the light, and that nystagmus is met with in greater frequency under such conditions. Other things, as nature of work, being equal, the disease will be most frequent with the bad light. Nieden and Dransart hold similar views. Romiée believes it to depend upon the efforts of accommodation induced by insufficient light, but it must be pointed out that the distance the miner is from his work is such that the accommodative effort can be but little if any.

"Nieden says that five per cent. of miners suffer. My observations support this generally; though in some parts the percentage is higher (I found among men working with candles that six out of a total of one hundred and forty were absent from work for nystagmus, and this represented only a portion of those who would have been found to be actually affected on an examination of the whole number). Romiée gives twenty per cent. for Belgium; possibly he includes less marked cases.

"The prognosis is good, and, even in old standing cases, if the directions as to work are followed the nystagmus will disappear. Treatment consists in a change of work. In some cases it will suffice if the patient ceases from coal-getting, without altogether stopping work in the mine, but generally it is advisable, especially if the nystagmus be of high degree and of some standing, to recommend cessation altogether of work under ground. Return to the mine is practicable after relief has been effected, provided the head can be kept straight and the upward turn of the eyes is avoided. Resumption of the old kind of employment is followed, sooner or later, by a recurrence of the symptoms. The patient's health will often be benefited by tonics, such as iron, and especially strychnia; in other cases the bromides are of service. Electricity has been used by some. Atropine and eserine, suggested by Romiée for their effect upon the accommodation, are of no use, nor is the condition they are supposed to benefit present. Tenotomy, formerly advocated, is useless.

"The affection has been observed by me, or brought under my notice by others, as occurring in compositors, a weaver, plank-cutter (Nieden), saw-maker, iron-founder, fitter, roller, plate-layer (railway), and a youth carrying a tray on his head."

Symptomatic nystagmus is observed in certain *vices of conformation of the head and of the brain* (Raehlmann), in *insular sclerosis* (Charcot), especially when the sclerotic isles affect the fourth ventricle and the optic thalami (Raehlmann), in hemorrhagic, embolic, or other lesions of the *optic thalami*, the *fourth ventricle*, the *restiform bodies*, and the *cerebellum* (Raehlmann, Hitzig), in partial infantile encephalitis, with secondary contraction of the *extremities* (Zehender). It may be consecutive to a traumatism (Nagel, Cohn, fracture by a fire-arm of the right temporal bone). At times it accompanies during the period of activity Cheyne-Stokes respiration (Merkel). Finally, Friedreich has pointed out as among the symptoms of hereditary tabes a peculiar form of nystagmus which he designates by the name of *ataxic*. The movements of the eyes seem then incoördinate, like

those of the lower extremities in the same malady. This interesting peculiarity has been found in several other cases of hereditary tabes described by other authors.

These few anatomo-pathological data can scarcely serve to elucidate the *etiology* and *pathogeny* of congenital and occupational nystagmus. Without doubt there must be made in this respect, as we have already said, a difference between these two forms. It has been sought to attribute congenital nystagmus to a change in contractility having a peculiar bearing on one of the ocular muscles. Boehm was of opinion that the internal rectus was at fault. This author distinguished between *tonic* and *atonic* nystagmus, or sometimes an excess of power, sometimes a weakness, of the internal rectus prejudiced the normal equilibrium of the muscles of the eyes.

It is probable that the diminution in visual acuity which so often accompanies congenital nystagmus is in intimate relation to this anomaly of vision. But one could not affirm whether both are the parallel effects of some central lesion (Raehlmann) or whether the imperfection of sight is really the cause of the nystagmus. Von Arlt, who holds this latter theory, believes that the oscillatory rhythmic movements are destined to increase the intensity of the insufficient visual impression made upon the retina by exterior objects. He bases this belief upon the fact that the object whose vision is rendered confused by a defect in optical adaptation, for instance, becomes more easily perceptible when it is shaken in front of the eyes. He also believes that the victims of nystagmus do not fix with the *fovea centralis*, as do persons whose eyes are normal, which maintains the constancy of fixation, but with some eccentric point in the vicinity of the posterior pole, so that the immobility of the gaze is indifferent. But this theory in no wise explains the compensatory movements of the head or the peculiar rhythm of the oscillatory movements, and it is contradicted by the occasional high degrees of visual acuity compatible with nystagmus (Alf. Graefe). We have already discussed the *rôle* which heredity may play in the development of this anomaly.

As to occupational nystagmus, attempts have been made to attribute it to peculiar toxic influences,[1] such as have been supposed to produce hemeralopia. But, apart from a more or less pronounced anæmia, no other phenomenon of chronic poisoning has been discovered among coal-miners, and, according to von Reuss, hemeralopia is lacking in many cases. Nevertheless, the same author has found nystagmus in the case of an inspector who did not take part in the occupation of the workmen in the pits, which fact would seem to argue in favor of an action produced by coal-gas upon the central nervous system.

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[1] This intoxication might be due to the various gases which are generated in the mines. We do not know any exact fact relative to the influence of these gases upon the oculomotor centres. As to other agencies being capable of an action of this sort, *eserine* must be cited, since it, in the case of Zehender's patient, provoked nystagmus.

However, the facts contributed by Magelson and Wilbrand, who have published cases of occupational nystagmus, which were altogether like that of miners, in seamstresses who are obliged to work with a poor light; and, on the other hand, the many cases in which nystagmus is associated with weakness or exaggerated working of certain muscles, the abnormal position which miners' eyes must have during work (asymmetric convergence with elevation of the gaze),—all these facts incline to the supposition that *fatigue of the muscles and exhaustion of their innervation* must exert a great influence favorable to the development of nystagmus. This is the opinion advanced by Baer, and upheld by Alf. Graefe, Wilbrand, Dransart, and Snell.

Every one is acquainted, indeed, with the chronic contractions presented by a muscle subjected to an exaggerated effort taking the place of the tonic contraction which is produced under the influence of the will when the muscle is not yet fatigued. It is highly probable that a similar phenomenon occurs in the ocular muscles in occupational nystagmus.

*Prognosis.*—Congenital nystagmus presents only rare cases of spontaneous cure.[1] As to occupational nystagmus, it is ordinarily curable in a shorter or longer time after the cessation of the existence of the cause by itself or under the influence of treatment.

The prognosis of nystagmus caused by central lesion is subordinate to that of the lesions themselves.

*Treatment.*—When congenital nystagmus is associated with a marked strabismus or with a refractive error, one may try to attenuate or suppress it by surgical and optical means. But congenital nystagmus almost always defies all kinds of treatment that may be directed against it.

As to occupational strabismus, we have nothing to add to what Dr. Simeon Snell has said above concerning its treatment.

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[1] One such case has been observed and reported by Alf. Graefe, loc. cit. p. 239.

# DISEASES OF THE CORNEA.

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TRANSLATED

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## INTRODUCTION.

DISEASES of the cornea constitute one of the most important chapters of ophthalmology, this being due to their serious consequences in regard to sight and their frequency.

So far as their own importance is concerned, they all disturb the transparency of the corneal tissue, so that if they invade the central or the pupillary zone they lessen the clearness of vision. Indeed, they but too frequently destroy all useful visual power. In this respect conjunctival ailments are less disastrous; they attack vision only in a secondary way, as it were, through their complication with corneal diseases.

As to the frequency and the importance of corneal disease, our statistics based upon the records of twenty thousand cases of affections of the eye show that from one-fourth to one-third of all ophthalmic disturbances consist in such diseases. Such is the proportion generally found. In countries where granular conjunctivitis is endemic, the relative frequency is somewhat less.

From observations made by Uhthoff on ten thousand blind people, it appears that in over thirteen per cent. of cases blindness is the consequence of corneal affections. If to these are added the cases in which blindness is consequent upon blennorrhœa (a result that is likewise produced by corneal complications), it will be found that in more than twenty-seven per cent. of cases—that is to say, in more than one case out of four—blindness is due to corneal affections (see vol. ii. p. 432 *et seq.*).

With reference to the frequency of corneal disturbances it is well to recall the fact that, first of all, this membrane is, of all portions of the eyeball, the most exposed to external injuries; and that, secondly, it is not supplied with blood-vessels. In addition, its different parts (especially the central zone) are quite a distance from the nutrient vessels, and hence,

to a certain degree, notably as compared with the conjunctiva, it lacks sufficient strength to combat the causes of disease. Nevertheless, clinical experience teaches that in the case of pathogenic influences that are not microbic, such as traumatic disturbances, for instance, the cornea can very well take care of itself. Its regenerative processes are almost as rapid as those of the conjunctiva, and are much more energetic than those of the fibrous tissues, such as the sclerotic.

As soon, however, as the reparative processes are complicated by microbes, the cornea displays a notable inferiority of regeneration in comparison with the conjunctiva. It seems that the cornea's relatively abundant stock of nutritive fluids and interstitial lymph is generally sufficient, as in the conjunctiva, for regeneration, when they are unimpeded by septic poisoning, to take place. If, however, there is pathogenic microbic complication, regeneration is delayed, and in too many instances the microbes gain the victory, this being much more frequently so than with similar changes in the conjunctiva. The reason of this is that the struggle against microbic influences is, above all, a function of the migratory cells (phagocytosis). In the cornea these cells frequently have a very long distance to extend before they reach the spot that is attacked by the enemy. In the cornea it is especially in the centre of the membrane that microbic infection develops and spreads the most easily.

All portions of the structure of the cornea are subject to the influence of ordinary pathogenic causes,—physical, chemical, and microbic,—but they are not equally susceptible to alteration of nutrition in consequence of such noxious influences. Bowman's membrane and the hyaline lamina of the membrane of Descemet are relatively slow to take on pathological processes. On the other hand, the epithelial and the endothelial layers react as energetically to pathogenetic causes as does the substance of the cornea proper. Alterations, however, that are limited to these membranes rarely produce changes that are sufficiently deep to injure vision seriously, and hence, unlike those of the substantia propria, can hardly merit the name of maladies in the clinical sense of the word. From this point of view, however, it seems that the *rôle* of the epithelium has in the past been too much neglected.

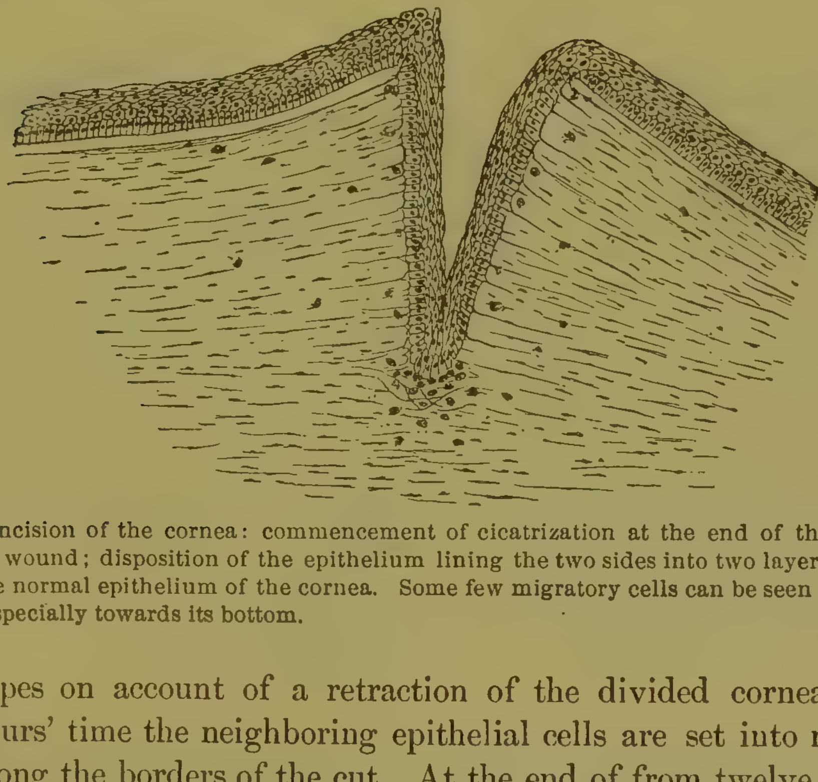
### A.—INFLAMMATION OF THE CORNEA. GENERAL PATHOLOGY.

Under the effect of various influences (traumatic, poisonous, etc.), the tissues of the cornea react against certain processes, which, when a sufficient intensity of reaction is attained, are designated by the generic term of "keratitis," or "inflammation of the cornea," this expression being borrowed from ancient theories. The manifest purpose of such inflammatory reactions is the repair of injury. The wounded elements are devitalized, are absorbed or are detached, and the resulting gaps are filled in, brought together, repaired, and cicatrized. When, in consequence of a superficial lesion of the corneal epithelium encroaching even but slightly on the substance

proper of the cornea, the neighboring epithelial cells alter their form, become active, and proliferate to fill the gap, this condition, however, is not, generally speaking, characterized as an inflammation. The line, nevertheless, at which the inflammation or keratitis in a clinical sense begins is not clearly traceable. In the example cited, there is certainly a keratitis, but the condition is of too little importance to be considered as such practically. The loss of substance once being regained, keratitis is no longer spoken of, although the cicatricial tissue may continue to change and to attempt to reapproach a normal state. Again, there is no distinctly traceable limit between corneal inflammation and the cicatrized tissue.

From a histological point of view, the various forms of keratitis can be especially noted by tracing them from the least to the most complex varieties. A simple case is that of an incised wound of the cornea which cicatrizes without the intervention of pathogenic microbes. The slight

Fig. 1.

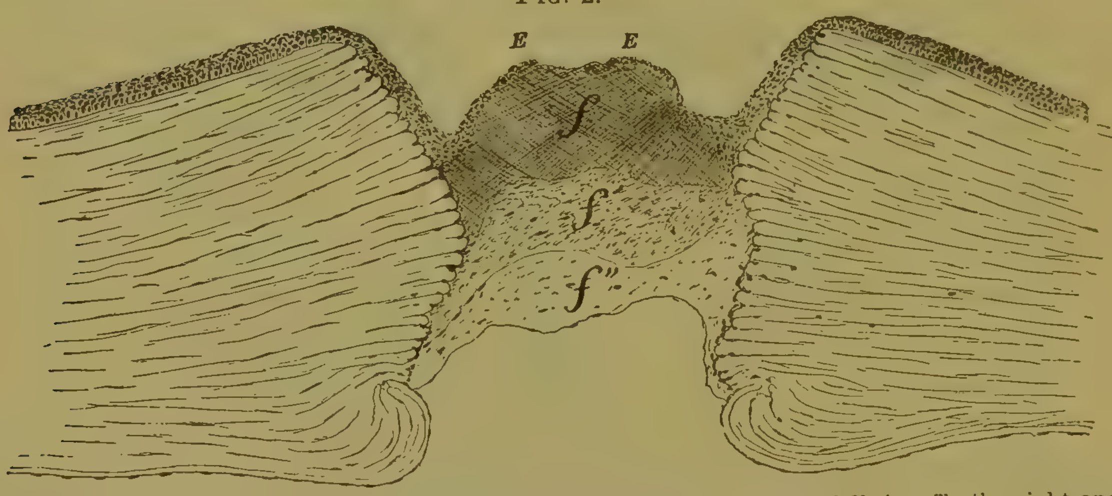


Vertical incision of the cornea: commencement of cicatrization at the end of the second day; gaping of the wound; disposition of the epithelium lining the two sides into two layers, as similar as possible to the normal epithelium of the cornea. Some few migratory cells can be seen in both lips of the wound, especially towards its bottom.

wound gapes on account of a retraction of the divided corneal lamellæ. In two hours' time the neighboring epithelial cells are set into motion and wander along the borders of the cut. At the end of from twelve to twenty-four hours' time they cover both borders of the incision, including even the bottom of the gap. (Fig. 1.) There is a multiplication of the epithelial cells by subdivision (karyokinesis), these being found partly in the wound and partly at a distance from the surface. Close by the incision the divided corneal lamellæ become swollen, the interfibrillary spaces distend (œdema), and after some hours, a few migratory cells appear. At times, the epithelial plug occupies more or less of the gap, starting to fill it in from the bottom. Always, however, the epithelial cells have a tendency to group themselves into two layers, one for each side of the wound. Finally, in consequence of the approach of the sides of the wound to one another, the epithelial plug is forced to the surface. The corpuscles of the cornea proliferate around the

incision, and the cicatrix of the tissue proper becomes marked by a moderate accumulation of young cells; these being transformed into fibrillary tissue, which for a long time, because of its irregularity, is discoverable under the microscope. The œdema disappears, and the epithelium, in the normal course of the healing process, follows in advance of the cicatrix. [If the incision is deeper the processes are more complex.] A common condition is that in which the incision has traversed the entire thickness of the cornea. If, under such circumstances, the wound does not gape too much, its two lips will come in contact by the median corneal planes. As a rule, the retraction is greatest before and behind, and at these points the tissues do not come in contact. The anterior interspace becomes filled in by the process above described. The posterior opening towards the anterior chamber is at first filled by a plug of fibrin which is furnished by the ciliary body and the iris. This plug is later infiltrated by migratory cells and transformed into cellular tissue, which is reconstituted into fibrillary material. The endothelium

FIG. 2.




Provisional occlusion of a corneal perforation (of a rabbit) by a plug of fibrin. To the right and the left are the two œdematous lips of the corneal wound; $f, f', f''$ are three successive layers of fibrin which have been successively formed. The wound is of two days' duration. Some few migratory cells appear on the lips of the wound, and in the two lately formed layers ($f', f''$) of fibrin. The epithelium has emigrated in front of the plug of fibrin, which it covers only as far as $E$ $E$.

slides over the plug, and later, by a process analogous to that which is occurring in the epithelium, it collects into a continuous layer. The emigration of the endothelium plays a less important *rôle* in the process of cicatrization. Behind, the interspace is filled by the insertion of fresh cellular tissue which is much more abundant than that which closes the wound in front. The membrane of Descemet does not always completely reform. The epithelium comes down little by little along the sides of the incision in a continuous layer of one or two cells, and in twenty-four hours' time a point is reached at which the adjoining sides of the wound touch, the layers of the two sides meeting one another. Emigration continuing, the cells accumulate at the bottom of the incision, filling it more or less, though rarely entirely. Henceforward, the tendency is to leave the central space free. The superficial cells become flattened, and the deeper ones assume a

cylindrical form which is perpendicular to the sides of the incision (as in the normal epithelium).

The epithelial cells multiply on the surface of the cornea as well as in the excavation. The œdema of the cornea extends more or less distantly. From the second day there is a rising in the tissue proper towards the edges of the wound, the migratory cells coming from the pericorneal vessels. There is, moreover, karyokinesis of the fixed corneal cells. The young cells of both productions penetrate beneath the immigrant epithelium, especially in the bottom. In this situation they constitute young cellular tissue which drives back the epithelium towards the surface, at the same time that the sides of the incision approach one other.

If the lips of the wound do not come in contact at any point, the plug of fibrin plays a more important *rôle*. At first it establishes a temporary closure (Fig. 2), after which the process of cicatrization becomes analogous to that which occurs in the preceding case. The iris may also be engaged

FIG. 3.


Provisional occlusion of a perforating wound of the cornea (of a rabbit) dating some eight hours. To the right and the left are the lips of the corneal wound. *J* shows the iris engaged in the corneal wound, to the lips of which it is united by fibrin; *F* is a thick plug of fibrin which has exuded from the iris. The corneal epithelium has begun to emigrate over the plug of fibrin, but only as far as *E E*. Some few migratory cells which have left the iris are penetrating the plug of fibrin.

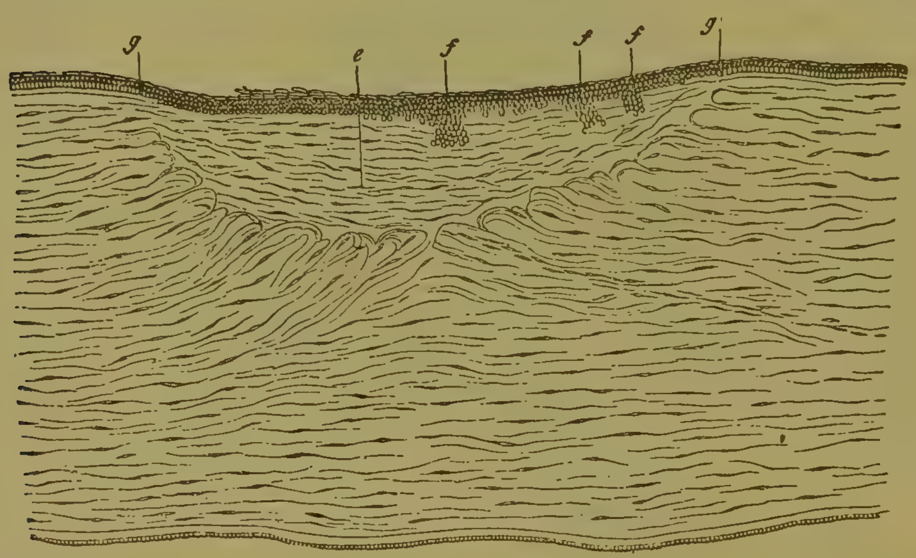
in the wound (Fig. 3), which it more or less fills. The iris is covered in front by a layer of fibrin, and the subsequent processes are virtually the same.—In the preceding cases the migratory cells have been furnished by the cornea; they have sprung from the pericorneal vessels. When, however, there is an attachment of the iris, its vessels become equally a source of migrating cells.—The iris establishes adhesions to the lips of the corneal wound. Its tissue rests fixed in the cicatrix, forming an anterior synechia.

As to the origin of this fibrin of the plug, it must be borne in mind that the normal aqueous humor contains hardly any albuminoid substances.

Re-formed after puncture of the corneal wall, albumin which is coagulable by heat and fibrinogenous substance are found in it. In the opinion of Greeff, the reason of this is that, after puncture, the epithelium of the ciliary processes rises up in vesicles. The tissues, thus stripped of their covering, allow the transudation of the plasma sanguinis which, in its normal condition, is held within the vascular channels by the epithelium; and it is this plasma that contains albumin and fibrinogen. Such, then, appears to be the origin of fibrin, consequent upon simple puncture of the cornea. In the frequent instances of corneal perforation through ulcerous keratitis associated with iritis and hypopyon, the iris assuredly supplies a portion of the fibrin that is contained in the anterior chamber. (See *Hypopyon.*) Besides, under these circumstances, the anterior surface of the iris is also affected; its endothelium is disturbed and even lost in spots. (In the case of Fig. 3, there is no doubt that the iris also is a source of fibrin.)

(*a*) *Cicatrization following Loss of Substance.*—Frequently there is a loss of substance, of greater or less extent in depth, on the surface of the cornea,

FIG. 4.




Cicatrized ulcer of the cornea: *f* represents the epithelium covering the cicatrix and penetrating it in the form of plugs; *g*, *g* designate the borders of the cicatrized ulcer, showing interruption of the membrane of Bowman; *e* signifies cicatricial tissue. (Saemisch.)

caused by a traumatism, as, for example, a cauterization. At other times the loss of substance occurs in consequence of an ulcer, in which case the bottom of the cavity is strongly infiltrated with migratory cells.—The cavity can no longer be obliterated by the approach of the lips of the wound to one another; it will become filled by cicatricial tissue. The repair commences by an emigration of the epithelium that lines the bottom of the cavity, followed by an accumulation of the young cells beneath it, which elevate it. In consequence of this the cicatricial tissue, which is at first cellular in character, becomes fibrillary in type and the cicatrix remains more or less opaque. (Fig. 4.)

Under the microscope the fibres are distinguishable from normal corneal tissue by a certain irregularity. Bowman's membrane is not reconstituted, the epithelial layer being irregularly limited towards the deepest part.

(*b*) *Corneal Infiltration.*—When, by means of a Pravaz syringe, a culture of staphylococcus is injected under the lamellæ of the cornea, the tissue involved becomes necrosed to a certain extent; migratory cells are brought in great number by the pericorneal vessels and collect at the edge of the necrosed tissues. One of their *rôles* is to act as a barrier to the wide-spread invasion of the microbes (phagocytosis). The corneal lamellæ become œdematous for some distance; they are softened; and at last are dissolved at the edge of the necrosed parts. It is assumed that the migratory cells finally conquer the microbes. The latter are digested and absorbed with the necrosed portions if they are not in too great numbers. It sometimes happens that the corneal layers lying above the injection are included in the necrosis; the whole is then detached and expelled, and the injured part becomes transformed into an open ulcer. In the end, repair is carried on by a similar process to that above described, with this difference, that the vicinity of the lesion is infiltrated by young migratory cells to a greater extent than it is in the repair of an aseptic lesion.

The term *infiltration* is given to the condition in which there is such an appearance in the normal elements of the cornea of those heterogeneous elements that more or less alter the transparency of the membrane. This infiltration material may be composed of a liquid (causing œdema), of precipitated substances, albuminoid matter (fibrin, etc.) in the form of granules, of microscopic filaments, and, finally, of cells.

Most frequently cells predominate in the infiltration mass. They are, however, always accompanied by œdema and by a granular precipitate. An example of cellular infiltration is that which is produced around an injection of staphylococci in the cornea to maintain it. One is disposed to admit nowadays, without sufficient reason, that all well-marked cellular infiltrations are the result of "microbic infection" through the penetration of pathogenic microbes into the tissue proper of the cornea.

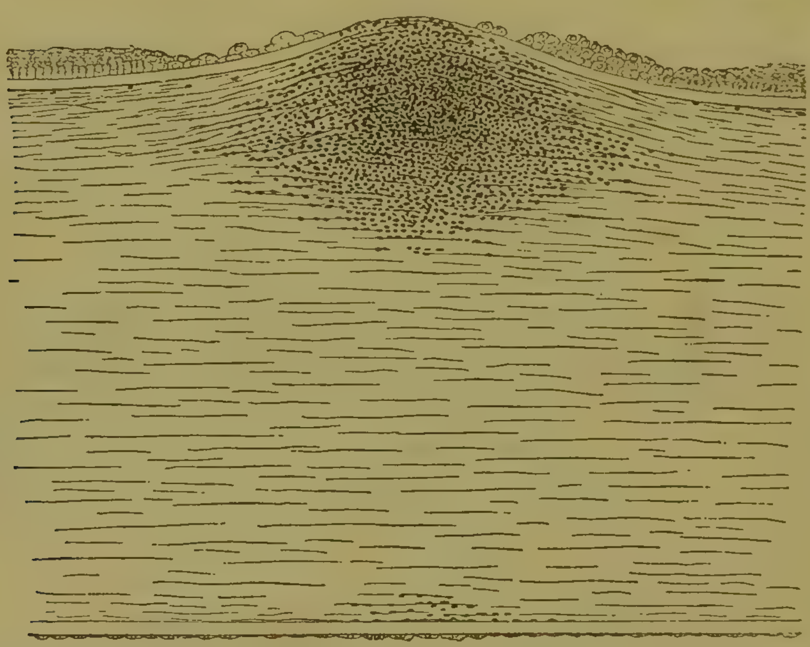
It is to a great extent dependent upon the results of experiments on the cornea that Metschnikoff founded his famous theory concerning phagocytosis. Since then, it has been generally admitted that the wellnigh exclusive purpose of cellular infiltration is to combat microbes. It is also admitted that phagocytosis and, consequently, cellular infiltration are the principal means of self-defence that the cornea puts forth against microbic invasion,—the bactericidal power of tears being too trifling to be taken seriously into account, any more than that of the aqueous humor in the case of corneal perforation.

It is unquestionably true that the penetration of pathogenic microbes into the tissues produces an immigration of young cells. It is likewise certain that, in the great majority of cases, cellular infiltration of the cornea is caused by the presence of these microbes. It does not, however, appear

certain that the process of combating microbes is the sole function of migratory cells. These cells seem to furnish a *dialytic* substance which softens dead tissues around the edges of healthy parts, and thus helps the unloosing of the dead portions (Leber). Finally, migratory cells contribute directly to the digestion, to the absorption, and to the removal of granular and other materials that have become unfit for the nutrition of the tissues (macrophagi). If, then, in the depth of the cornea there is deposited a substance—whether of an irritating nature or not—that is safe from microbic infection; if in consequence of disturbances in the general nutrition, nutritive anomalies, as yet ill-defined, manifest themselves in the cornea, in each and all of these conditions, migratory cells will speedily make their way (although in less quantity than in microbic infection) towards the affected spot, like so many ants hastening to their prey, in order to contribute to the absorption of the anomalous deposits and of the affected corneal elements.

The corneal infiltration may be superficial, involving only the epithelium, or is deep from the first onset. (Fig. 5.) Cellular infiltration is always

Fig. 5.



Superficial infiltration of the cornea, producing a projection on the surface of the cornea. At its level, the epithelium is exfoliated or is even absent. The infiltration approaches transformation into an ulcer. Behind, there are some migratory cells at its level, against the membrane of Descemet. The endothelium is slightly altered.

accompanied by œdema, the infiltrated portion being more or less clouded, according to its density. The epithelium is always exfoliated at its level, this being more pronounced as the infiltration is the more superficial. This exfoliation, clinically, is indicated by a dulness of the corneal surface.

There are diffuse forms of infiltration (see *Parenchymatous Keratitis*), and other varieties that are more circumscribed. A circumscribed form of superficial infiltration may produce a bosselated appearance of the corneal surface. (Fig. 5.)

If the infiltration is not too dense, it may be absorbed without leaving an appreciable trace; otherwise a cicatrix, which appears as a permanent macula, remains. When the infiltration is more notable in extent and in intensity, the infiltrated corneal tissue softens or becomes necrosed. In such a case it is preferably known as *suppurative keratitis.* This form always appears to be due to a microbic infection, and the infiltration, extending, reaches the epithelium. Bowman's membrane is uniformly softened, the epithelium is raised, and the infiltration area changes to an ulcer, the fundus of which is completely occupied by cells. (Fig. 6.)

FIG. 6.

Infiltrated corneal ulcer. (After Alt.) Œdematous corneal lamellæ curved towards the surface of the ulcer. The epithelium is about to descend into the ulcerous area.

(*c*) *Corneal Ulcer.*—Such an ulcer is always infected,—that is to say, pathogenic microbes play an important *rôle* in its evolution.

It happens in a well-formed ulcer that the infiltration ceases to extend, and repair begins. At other times, when the microbic infection is pronounced, the ulcer invades healthy corneal tissue and becomes deeper and broader (progressive ulcer). Sooner or later, however, the essential attributes or properties of the tissues (phagocytosis, etc.) conquer the pathogenic microbes, opposing to them an insurmountable barrier of cells,—a zone of infiltration. This process produces an arrest of the ulcer in which the mass becomes regressive, and cicatrization proceeds as described above.

In every case of well-marked keratitis, especially in the suppurative form, the area of focus affected is surrounded by a collateral œdema. There is congestion of the nutrient vessels of the cornea, and often of the vessels of the iris.

(*d*) *Microbic Infection. Exogenous and Endogenous Microbic Infection.*—According to the chemical theory of inflammation in general, and especially of microbic inflammation, the intra-corneal pathogenic microbes produce *phlogogenic substances, toxines,* the chemical nature of which is but little known; these are diffused in every direction, particularly towards the periphery of the cornea and in the anterior chamber. In the first direction they reach the pericorneal vessels, which they paralyze and whose walls they alter, and thus provoke exudation of plasma and emigration of leucocytes, which are attracted by these substances towards the region of infection, and constitute corneal infiltration. In the direction of the anterior chamber, if the toxines are sufficiently numerous, they reach the iris, where they cause the same vascular alteration, exudation of plasma, and emigra-

tion of cells,—*i.e.*, iritis. The exudation, especially the cells, may accumulate on the bottom of the anterior chamber under the name of *hypopyon*. These substances alone, isolated from the microbes and inoculated in the cornea (Th. Leber), are sufficient to provoke all the symptoms of microbic inflammation.[1]

As said above, it is supposed also that in all changes in the normal nutrition of the human tissues,—for example, in consequence of wounds or as the result of deposits of some heterogeneous principle,—there are produced phlogogenous substances which are analogous to, if not identical with, those of pathogenic microbes.

Microbic infection, the principal cause of cellular infiltration of the cornea, is nearly always exogenous in type. The microbes penetrate the corneal tissue proper through lesions that are sometimes purely epithelial in character. When the epithelium is intact, it presents an almost insurmountable barrier to them. It may be said that in all cases of ulcerous keratitis, whether it be superficial or deeply seated, if there is microbic infection—and that is always the case, either primarily or secondarily—it is exogenous in type.

Most frequently the infecting microbes in the exogenous form of infection are the staphylococcus aureus and the staphylococcus albus; often the diplococcus of Fränkel or pneumococcus (Gasparini, Uhthoff, and Axenfeld); and, less often, the more virulent streptococcus. Imperfectly described bacilli have been found in a few cases of this kind.

*Endogenous* microbic infection is very exceptional. Some microbes, coming from the pericorneal blood-vessels, after penetrating the subconjunctival tissue, might perchance pass into the cornea by means of the interstitial lymph-spaces. This form of infection seems to be established in the case of tuberculous parenchymatous keratitis, and perhaps also in that of leprosy. But the last case is a variety of exogenous infection, the bacilli of leprosy penetrating the pericorneal tissue directly, instead of being conveyed by the blood-current.

In the more frequent cases of syphilitic parenchymatous keratitis, it is not known whether the poisonous substance which penetrates the deep lamellæ of the cornea—always brought into position by the blood-currents—is a microbe or a chemical substance. According to all appearance, noxious chemical substances that have been conveyed by the blood-currents or formed in the pericorneal tissue and have penetrated the cornea are the cause of certain forms of deeply-seated, non-ulcerous keratitis (in gout, for instance).

A second form of endogenous infection would be that in which microbes, coming from the depths of the eye by means of the aqueous humor, would

[1] To give an idea of the attraction that is exercised from afar by phlogogens over leucocytes, it may be recalled that the antherozoids of ferns are attracted by malic acid, those of moss by sugar of milk, and mobile bacteria by peptone; and that the one-trillionth part of a milligramme is sufficient to produce this effect. (Pfeffer.)

penetrate the cornea through its posterior surface. The certainty of this method of corneal infection has not been established beyond question. On the contrary, what seems a rather frequent occurrence is that, in certain deeply seated affections of the eye (cyclitis, for example, and perhaps also scleritis), substances that are more or less noxious, but are not microbic in character, pass into and irritate the deeper layers of the cornea, including the endothelium, and produce localized cellular infiltration.

Formerly—and the practice still holds nowadays—simple *non-infected* ulcers were discriminated from *infected* and *suppurative* ulcers of the cornea. From a clinical point of view this point will be continued, but it must be borne in mind that in the former variety (the non-infected) there is infection through pathogenic microbes, although the infection is very trifling either because the microbes are less virulent or less numerous, or because the bactericidal power of the cornea is greater. On the other hand, by the term "infected ulcers" will be designated those cases in which a greater progressive infection is differentiated from a stronger local accumulation of young cells, and a more intense distant reaction (ciliary injection and iridian phenomena). Lastly, the term "suppurative ulcers" will be used when the accumulation of cells in the ulcer is very great, and especially when there is pus in the anterior chamber.

(*e*) *Formation of Vessels in the Cornea; Pannus.*—The regression of somewhat extensive corneal infiltrations and of ulcers of notable dimension is marked by the appearance in the corneal tissue proper of vessels which later disappear. In such cases this condition is a phenomenon of good omen; it strengthens the nutrition of the cornea, assists in the resorption of the exudations, and helps in the struggle against the invading microbes. Nevertheless, there are certain forms of superficial cellular infiltration that are accompanied by a vascularization which proceeds from the superficial pericorneal and even from the conjunctival vessels. This variety exhibits a tendency neither to disappear nor to suppurate, but tends to become definitely organized. Such vascularization, in some measure made an integral part of the pathological process, is not a favorable symptom. The name of *pannus* is given to these forms of superficial cellular infiltration and vascularization. (See *Phlyctenular Keratitis,* and especially *Granular Pannus.*) At first the vascularization is situated under Bowman's membrane, which it later pierces and penetrates beneath the epithelium. In deep keratitis (parenchymatosa) the vessels proceed from the deep pericorneal vessels and are situated in the deep lamellæ of the cornea.

(*f*) *Corneal Œdema.*—Corneal inflammations, traumatic, microbic, etc., are always accompanied by an œdema which extends more or less beyond the portion that is injured, infiltrated, or infected by microbes. The œdematous part is swollen and thickened. Clinical examination shows a diffuse disturbance that is uniform and smoky in appearance. Sometimes it is more intense in portions and even appears striated (dilatations of the interlamellar spaces). The corneal surface at its level is slightly dulled.

Microscopically, the interlamellar spaces are seen to be distended and the lamellæ are held apart by a clear liquid. Some migratory cells may accumulate in the œdematous parts, but they disappear before the œdema passes away. The disturbance is not due to their presence. Liquid may infiltrate between the epithelial cells, distending the intercellular lacunæ, while the epithelium may frequently irregularly exfoliate. In consequence, the cornea becomes turbid, or the liquid may elevate localized areas of the epithelium in the form of vesicles. The œdema surrounding a traumatic keratitis is partly a product of inflammation and partly an infiltration by tears and aqueous humor through the wound. The simple removal of the corneal epithelium or endothelium gives access to liquids (aqueous and tears) overflowing it, which impregnate and disturb the corneal tissue (Leber). Non-inflammatory œdema due exclusively to this cause will be met with. The corneal œdema of acute glaucoma appears to result from a stasis of the corneal lymph. The corneal œdema of superficial punctate keratitis, being wholly inflammatory, is unaccompanied by cellular infiltration; it is an inflammatory form of lymphatic stasis.

## B.—CLINICAL EXAMINATION OF THE CORNEA. SYMPTOMS OF KERATITIS.

To expose the cornea the fingers of one hand ordinarily suffice. The employment of cocaine is recommended as a general measure to facilitate exploration. Exceptionally (as in extreme swelling of the conjunctiva, violent blepharospasm, etc.) it becomes obligatory to use the hook-shaped retractor of Desmarres. Chloroform narcosis is never necessary.

The treatment of an ocular malady in general should never be commenced without having the cornea, the conjunctiva, the anterior chamber, and the iris well examined. Not only should this be done on account of the responsibility involved, but also to have the value of the significance of the symptoms that have been determined more thoroughly understood.

**General Clinical Symptoms of Keratitis.**—In order to have the condition known as keratitis, it is necessary to have the following three pathognomonic symptoms: 1, corneal disturbance; 2, inequalities or dulness of the corneal surface; and 3, ciliary injection.

1. *Corneal Disturbance.*—Gray or yellow discoloration in varying degrees may be present. Simple epithelial lesions produce this discoloration only to an insignificant degree. If it is trifling, the naked eye cannot observe it, especially if the iris is clear. When the eye is directed so that the spot is projected in front of the black pupil, the disturbed area becomes more plainly visible. Oblique, focal, or lateral illumination (see *Special Article*, vol. ii. p. 27) will decide in difficult cases when other symptoms give rise to suspicion of keratitis. If the disturbance is very circumscribed or superficial, a presbyopic observer should provide himself with a convex lens of two to twenty diopters. If he wishes, he may use a Bruecke's loupe. The binocular loop of Zehender, etc., and the corneal microscope (see vol. ii. p. 27) have also been employed to advantage.

The use of these is complicated, and should be reserved for researches that are special in character.

The appearance of inflammatory disturbance may be profitably intensified or even made quite discernible by coloring matter that is made to penetrate the corneal tissues through epithelial lesions. A drop of a two per cent. strength aqueous solution of fluorescine gives a greenish-yellow color to the substance proper of the cornea, and resorcine, in two per cent. strength aqueous solutions causes it to assume a reddish-brown color. In this manner an exact idea of a corneal lesion—for example, a superficial abrasion—can be formed. Old corneal spots and maculæ are not colored, nor are deep corneal œdemas or deep infiltrations, if the epithelium is intact. This is also the case when there is simple deposit on the posterior surface of the cornea.

2. *Dulness or Inequalities of the Corneal Surface.*—A simple dulness in the plane of an inflammatory infiltration, and even before any corneal œdema reaches the surface, may only be seen directly by binocular vision. This is also true when there are slight excavations, ulcers, etc., of the corneal surface. This in a measure can be determined by observing a regularly formed image made by reflection on the corneal surface. To do this, the eye to be examined should be placed a little in profile before a window, when, if the corneal reflection is normal, a small plain image of the window may be recognized. If there is an irregularity of the surface, the straight lines of the reflected images are diffused, etc. In the case of ulcer or of marked elevation in the cornea, the image of the window is scarcely recognized, but the configuration of the cornea is determined. By means of the moving finger the eye examined should be directed in such manner as to bring successively the different corneal segments to any point in which the corneal reflection is found. A slight dulness may be obscured by the tears, this condition reappearing if the eye be kept open for a brief period of time.

3. *Ciliary Injection.*—The cornea is nourished exclusively by the pericorneal vessels, both superficial and deep, which are subdivisions of the anterior ciliary vessels. The superficial ones preside rather over the nutrition of the anterior corneal planes, the corneal conjunctiva, and the limbus conjunctivalis, while the deep ones control the circulation of the deeper planes. In every fairly marked case of keratitis both sets of vessels are congested, but the superficial alone are visible—although the trunks are common to both. The blood of the ciliary veins is red, just as is that of the conjunctival veins, this being caused by hæmatosis from contact with air passing through the conjunctiva. The nourishing superficial capillaries of the cornea are found in the limbus conjunctivalis. Capillary injection constitutes, accordingly, a roseate band that is situated around the cornea. It is general, if the keratitis is central or very extensive, and it is localized or very marked at some particular point, if the keratitis is peripheral. Frequently, the conjunctival vessels are dilated and conceal more or less the ciliary injection (collateral hyperæmia). In fact, there can be a conjunctival

catarrh, which may be the cause or the consequence of a keratitis. It is important to distinguish between the two forms of injection, the inflammation of the cornea being generally the more important and the one demanding special treatment. (For conjunctival injection see *Diseases of the Conjunctiva.*) Some of the differential points are, (*a*) injection of the ciliary capillaries reaches its maximum near the cornea, and diminishes towards the equator of the eye, as opposed to conjunctival capillary injection; (*b*) the injection of the ciliary capillaries is frequently unequal or partial, while the conjunctival is general; (*c*) the large ciliary trunks lose themselves in the deeper parts of the sclera before they arrive at the cul-de-sacs; (*d*) the ciliary vessels run serpentinely without giving origin to any large collateral branches, while the conjunctival ones subdivide in an arborescent form; (*e*) the conjunctival vessels distribute themselves only in the conjunctiva; (*f*) the conjunctival vessels are of a cinnabar-red color, while the ciliary ones are violet in tint. (*g*) The former can be displaced laterally by incurving the conjunctiva; the latter are fixed and traverse the sclera in regular grooves. In chronic glaucoma, for example, the ciliary trunks alone are injected. The pericorneal capillaries are injected in diseases of the cornea, of the iris, and of the ciliary body.

**Symptoms of Relative Gravity of Keratitis.**—It is essential to observe all the symptoms of keratitis. Among them, however, there are some which denote an exceptional gravity of the malady,—that is to say, the danger of harmful infection and complication with iritis is sufficient to render necessary a special form of treatment, such as atropine, antiseptics, and occlusive bandages. A keratitis is always grave, and demands particular care, first, when it is pronounced and involves the deep corneal layers, and, second, when it is strongly infected. In the latter case the phlogogenic substances are diffused in the deep corneal planes and much further (in the aqueous humor and in the iris), provoking the symptoms of profound keratitis. Direct inspection of the corneal lesions is not always sufficient to decide if they involve the deep corneal planes or if the structures are infected. The intensity of an infiltration is not an absolute criterion of the degree of the infection. A profound one may be concealed by a superficial disturbance. As a rule, however, it is difficult to determine the level of any form of such infiltration. The corneal lamellæ of the bottom of a deep ulcer may perhaps be swollen and project even to the surface, etc.

As it is, therefore, seldom possible to ascertain by direct inspection the relative gravity, the depth or the degree of infection of a case of keratitis, it is of importance to pay attention to the following symptoms, which always reveal the gravity of the disease.

These symptoms are: 1, congestion of the iris; 2, intolerance of irritating drugs; and, 3, the formation of deeply-seated vessels in the cornea.

1. *Symptoms of Congestion of the Iris.*—The cases of profound keratitis, of aseptic or septic infiltration, and of deep aseptic wounds, as well as those that are manifestly infected, are always accompanied by a group of iris

symptoms, which most frequently are due to congestion of the iris tissues. In extreme cases the dilated vessels of the iris can be seen, but this is not generally so; so that it becomes necessary to judge of this by indirect means. These symptoms of congestion of the iris are the following: (*a*) contraction of the pupil; (*b*) sluggish action of the iris; (*c*) discoloration of the iris; and (*d*) ciliary pain (see *Diseases of the Iris*). When posterior synechia is added there is iritis, which is characteristic of serious forms of keratitis. The greater part of these symptoms, with the exception of the discoloration of the iris and synechia, may give rise to a spasm of the sphincter muscle of the pupil. Spasmodic myosis is also accompanied by ciliary pain. The condition is also observed in cases of somewhat extensive superficial keratitis, being caused by irritation of the corneal nerves. If the affection is situated even moderately deep or if it is infected, it will be complicated by a true hyperæmia of the iris. In profound or in infected cases the myosis is at the first onset congestive in type. It is a collateral œdema that is capable of progressing to inflammation, this result probably being dependent upon a diffusion of phlogogenic substances from the cornea towards the iris. Dilatation of the vessels of the iris distends the membrane and also contracts the pupil.

Ciliary pains result from a twitching of the nerves of the iris. They cease as soon as atropine dilatation of the pupil is reached, but if atropine fails to dilate the pupil, etc., they continue in consequence of synechia. They radiate over the forehead and even extend to the cheek, being very characteristic of a profound keratitis. It is necessary to distinguish them from so-called superficial pains, which consist of sensations of scalding, of heat, and of foreign bodies located in the eye or in the eyelid; these being mainly observed in cases of superficial keratitis, and proceeding under irritation of the corneal nerves. Ciliary pain is intensified at night and is influenced by atropine, while superficial pain yields to cocaine.

2. *Intolerance of the Eye with Respect to Irritant Medicaments.*—In many cases the gravity of a keratitis is shown by the ulcer extending in depth, or becoming strongly infected, by the effect exhibited from the use of irritant remedies employed in the treatment of the superficial forms of inflammation, such as seen in phlyctenular or granular disease. These agents, far from acting favorably, exaggerate the symptoms of irritation, such as ciliary pain and injection, for an hour or more. Warning will thus be given to discontinue any irritant medication, and an opportunity given to apply the treatment that is necessary for deep keratitis.

3. *Deep Vascularization of the Cornea.*—Where a keratitis is complicated by vascularization of the cornea, the affection is seen to be profound or superficial in accordance with the situation of the vessels, this often being determined by inspection when such vessels traverse a portion of the cornea that is still more or less transparent. It is especially important, in the profound forms of keratitis, to examine the eyes simultaneously with the ophthalmoscope and with a convex lens of from eight to twenty diopters

(see vol. ii. p. 56). Ordinary binocular vision, however, is sufficient to determine the level of the vessels.

| SUPERFICIAL CORNEAL VESSELS. | DEEP CORNEAL VESSELS. |
|---|---|
| May be traced as far as the episclerotic ciliary vessels. | Disappear under the limbus conjunctivæ. |
| Are easily observed. | Are less discernible on account of their depth. |
| Subdivide like the branches of a tree, and are often sinuous. | Are seldom subdivided; are parallel to one another. |
| May elevate the anterior level of the cornea, producing a surface which is raised and bosselated. | The corneal surface is regular, not elevated by the vessels, but dull. |

**Other Symptoms of Keratitis.**—4. *Photophobia and Blepharospasm. Lacrymation.*—Photophobia, pain caused by the impinging of light upon the eye, is scarcely explicable physiologically. It is known, however, that light does produce a painful impression, but it is impossible to explain satisfactorily its *modus operandi.* Blepharospasm, cramp of the orbicularis muscle, is partly a result of the photophobia and partly a reflex spasm, which is produced by irritation of the corneal nerves. Like dread of light, it is observed even in cases of superficial keratitis.

From a knowledge of the special functions possessed by nerves, the phenomenon of photophobia is an extraordinary one. Evidently the sensory fibres of the cornea are not excited by light. It would, therefore, seem to follow that the impression of light upon the retina is painful, which is in opposition to the theory that retinal excitation can produce any other sensation than that of light. The matter is capable of being understood, if it is admitted that the pain is not strictly connected with special afferent nerves, but that it results from an excessive excitation of any centripetal nerves that are present. This is probably the true explanation. Thus, in section of a non-atrophic optic nerve, the subject operated upon will complain of a sensation of light that may be so severe as to be "painful." Again, traumatic luxation of the eyeball in front of the orbit produces a painful sensation of light which is insupportable, the pain being somewhat similar to that which is observed when attempts are made to look at the sun with the naked eye. The theory is, therefore, advanced that in cases of keratitis, accompanied by photophobia, a superficial inflammation of the sensory nerves of the cornea provokes a hyperæsthesia of the optic nerve, or rather of its central termination, just as ordinary light gives rise to a painful sensation in the optic-nerve apparatus. In this connection it should be mentioned that inflammation of the retina and of the optic nerve do not produce true photophobia.

The explanation of the phenomenon of blepharospasm is more comprehensible, its purpose being to guard the hyperæsthetic visual apparatus from light, which is functionally painful to it. A different kind of spasm of the orbicular muscle may be observed when the eye is not at all irritated.

This condition, when not attended with pain, is known as *tic non-douloureux*, and is believed to be due to a lesion that is situated either in the facial nerve or in its nucleus. At other times the spasms are painful (*tic douloureux*), and result from an excitation of one or more branches of the trigeminus outside of the ocular globe, such as dental caries, or inflammation of one of its branches or of the Gasserian ganglion. The contractions seen in *tic douloureux* seem always to be of a reflex nature. In true blepharospasm due to corneal disease the spasm is provoked both through the optic nerve and through the corneal branches of the trigeminus. Cocaine, by anæsthesia of the nerves of the cornea, sometimes causes it to disappear; again, it seems only to diminish it. The spasm rarely disappears even in the dark.

Reflex sneezing, which so frequently occurs in diseases of the cornea accompanied by blepharospasm, is likewise provoked by a combined irritation of the corneal nerves and of the retina. Indeed, many persons in good health sneeze when they gaze at a focus of light. In corneal disease, sneezing is induced when the eyelids are violently opened, more especially, when they are held apart for some time. As the act of sneezing is not suppressed in the dark, it is proper to suppose that the morbid condition which incites it is due to the prolonged contact of the air with the cornea, which is painful even in the normal state. In other respects, true blepharospasm is not so purely a reflex phenomenon as in *tic douloureux* or *tic non-douloureux*. In the beginning, at least, the contractions of the orbicularis muscle are more or less intentional, being designed to relieve the photophobia. Even in the later stages the will is able to moderate them. As opposed to *tic non-douloureux*, blepharospasm must, therefore, be regarded as half voluntary and half reflex, and an act which is always more or less under the control of the will. Horner has maintained that blepharospasm is exclusively caused by the patient remaining in the dark, and states that twitching does not appear if they are forced to live in well-illuminated places from the time of the beginning of the disease. This opinion of Horner is too dogmatic, although the writer does not deny that living in the dark is of a nature in itself which tends to exaggerate any existing blepharospasm that has been induced by retinal hyperæsthesia.

The lacrymation, which occurs so frequently in cases of keratitis that are accompanied by pain and by photophobia, is another reflex phenomenon which is due to irritation of the sensory nerves of the cornea: the flow of tears is induced by a reflex innervation of the sensory nerve-fibres of the lacrymal gland.

5. *Visual Disorders.*—When an eye is first affected by a keratitis it quickly becomes fatigued. Should corneal alteration or cellular infiltration and œdema appear and extend in front of the pupil, visual acuity will be diminished. (For the mechanism of this form of disturbance of vision see chapters on *Maculæ Corneæ.*)

6. *Tension of the Eye.*—In moderately extensive keratitis, which has injured both the epithelium and the endothelium, intra-ocular tension becomes

reduced, for the reason that elevation of the endothelium particularly favors filtration of the aqueous humor through the corneal tissues. Intra-ocular tension becomes nearly abolished whenever an ulcer has perforated the cornea.

7. *Sensibility of the Cornea.*—This condition, tested by means of a filament of cotton, may be diminished. At times this diminution is in a measure dependent upon paralysis of the trigeminus, and may be either the cause or the effect of the corneal inflammation. In cases of extensive and severe keratitis, diminished sensibility of the cornea may be easily produced, and may coexist with ocular and periorbital pain.

### C.—GENERAL THERAPEUTICS.

In the treatment of keratitis the primary object, although a negative one, is to avoid irritating the cornea, as corneal diseases generally are aggravated by irritants, and subside when irritating influences are removed. This is one of the most important principles in the treatment of diseases of the cornea.

A frequent cause of irritation lies in the use of medicinal substances (ointments, collyria, etc.) which the physician is but too often tempted to apply to the surface of an eye that is affected with keratitis, especially when the inflammation is accompanied by a secretion. As a general rule, such drugs should be employed only in exceptional cases, which will be duly specified.

1. *Dressings of the Eye: the Occlusive and Compress Bandage.*—A common cause of mechanical irritation is the act of winking. By rubbing the affected parts, which, as a rule, are more or less ulcerated, the eyelid irritates them and disturbs the histological processes of regeneration. For the same reason that ulcers of the skin should be covered with a piece of plaster the affected cornea should be protected against the rubbing of the eyelid, and this cannot be better done than by an occlusive bandage. It is therefore necessary to immobilize the eyelids by a well-applied bandage. This can be done so as to exert a certain amount of pressure on them, especially towards the nose, on a level with the tendon of the orbicularis muscle, by a padding made of some elastic material, such as cotton. To render the bandage more absorbent of the tears, conjunctival secretions, etc., a pad of gauze should be placed over the eye, the periorbital hollows being filled in by cotton that is held accurately in place by the bandage. It is not necessary to employ the so-called antiseptic cotton and gauze; scoured cotton is sufficiently aseptic, or it and the gauze can be rendered so by steaming. Impregnation of both cotton and gauze with antiseptic liquids impairs the absorbent power of the dressing. In fact, a non-aseptic but absorbent dressing is of greater value than an antiseptic but non-absorbent bandage.

Where perforation of an ulcer is threatened, or when it has actually occurred, the bandage should be applied more tightly, so as to compress the cornea. The bandage should be renewed at least daily, and even more fre-

quently if there is any lacrymal or conjunctival secretion, especially of the muco-purulent type, which when retained in the eye becomes a culture medium for bacteria. If the conjunctival secretion is very profuse, as in the blennorrhœic and diphtheritic forms of conjunctivitis, or when there are exuberant granulations, bandages may be harmful; the conjunctival disease is then usually the *cause* of the keratitis. Bandages, however, are useful when the conjunctival secretion, even though slightly purulent, is a consequence of an ulcerous or a suppurative form of corneal inflammation. Finally, there are cases of simple keratitis in which the bandage is harmful, or at least it is useless; these exceptions will be described later on.

The bandage often acts as a preventive for the entrance of dust particles, whether they be infected or not. Normally, by collecting such foreign substances on the free borders of the eyelids and by driving them towards the lacrymal ducts, the movements of the eyelids are sufficient to expel them from the polished surface of the cornea. Such is not the case, however, when the corneal surface is roughened and ulcerated; the particles of dust, microbes, etc., collecting in the depressions and on the borders of the eyelids cannot be removed by the act of winking. In keratitis, the movement of the upper lid is like that of a pencil loaded with a culture of some pathogenic microbe by which the cornea becomes inoculated. From this it can be understood that immobilization of the upper lid by a bandage is the means, *par excellence,* for avoiding infection of the cornea from any irregularities of its surface, as well as for the purpose of maintaining cleanliness.

2. *Antiseptics.*—Most cases of keratitis are infected or are liable to become so through even the slightest break in the epithelium. Hence it is always necessary to employ antiseptic measures,—that is, to use well-devised means of cleanliness. It is necessary to cleanse thoroughly by means of antiseptic solutions, not only the surface of the cornea and the conjunctival sac, but also, and above all, the free edge of the eyelids (that receptacle for microbes) and the surrounding skin. While absolute asepsis of the surface of the eye is impossible, the number of microbes may be so diminished that they are unable to exert any injurious action, thus allowing the bactericidal properties of the tissues to acquire the mastery over them.

In healing this condition various antiseptics are employed, but, unfortunately, two of the best of these—carbolic acid and nitrate of silver—are too irritating to be of any value in the treatment of keratitis. Preference should, therefore, be given to the choice of those antiseptics that possess as few irritating properties as possible. The best of these are the soluble salts of mercury, which are most often employed in the form of an aqueous solution of the bichloride in the strength of 1 to 2000, 1 to 5000, and best 1 to 10,000. In using this solution the ocular surface of the conjunctival sac should be flooded and a dry bandage applied. In the case of deeply infected ulcers, compresses wet with a solution of 1 to 5000 can be placed over the eye, the action of the drug in this strength being less intense but more lasting. Cyanide of mercury in aqueous solution of 1 to 5000 is said to be less irri-

tating (Chibret). Biniodide of mercury in the proportion of 1 to 20,000 is also employed (Panas). Iodoform in powder, followed by the use of a bandage, is less energetic, but its action is more enduring, diminishing the conjunctival secretion, particularly in cases of infected ulcers. This drug acts as an irritant to an eye that has just been flushed with bichloride of mercury solution, forming therewith an iodide of mercury. In some cases pyoktanin may be recommended as an antiseptic of value (Stilling). This drug appears in the form of different aniline colors, the methyl blue being the one that is preferred. It should be applied as an aqueous solution varying in strength from 1 to 1000 to 1 to 10,000. Methyl blue may a'so be used in the form of a crayon, with which the ulcer can be touched after cocainization. The tincture of iodine applied with a camel's-hair brush to the ulcers could be employed to advantage, were it not for the extreme pain arising from its use, even in those cases in which previous cocainization of the eye has been practised. Chlorine water has also been recommended for the treatment of this condition. Applications of protargol do not seem to be attended with the same beneficial results as when they are used in blennorrhœic conjunctivitis. Under keratitis reference will be made to the use of subconjunctival injections of the salts of mercury and of the cyanide of potassium.

3. *Cauterization.*—The actual cautery has been brought into general use chiefly by Gayet, and at the present time is employed in the form of the galvano-cautery. See Vol. III. of this System, p. 823. It is efficacious in the treatment of infiltrations and infected progressive ulcers, even when there is hypopyon; it destroys the most intense varieties of corneal infiltration and the nests of microbes, while other complications, such as œdema of the cornea, iritis, and hypopyon, that are ordinarily the effects of remote microbic action, improve rapidly under this form of treatment.

4. *Mydriatics.*—Sulphate of atropine is employed in the form of collyria of one or two per cent. strength, made aseptic by the addition of corrosive sublimate. It is a remedy of great utility in many cases of keratitis, its use being indicated whenever there is an iritis or even a congestion of the iris. By dilating the pupil, it contracts the vessels of the iris, and thus has practically an antiphlogistic effect, while ciliary pain subsides as soon as dilatation commences. Ordinarily, the corneal inflammation itself improves rapidly, leading many to suppose that the drug acts favorably on the corneal lesion also. This, however, does not seem to be the case, although it is doubtless true that any congestion of the iris acts unfavorably on a keratitis. A single drop of atropine dilates the normal pupil, but when there is congestion of the iris repeated instillations (made by the physician himself) become necessary, the attendant maintaining any mydriasis that may have been secured by the medical adviser. In order to secure good results, it often becomes necessary to make frequently repeated instillations. Application of contaminated atropine may produce pain, which, however, may be promptly relieved by the use of a drop or two of cocaine.

During the instillation the canaliculi should be compressed, and care should be taken not to drop any of the drug into the mouth. When the drops are properly introduced into the eye atropine-poisoning is not greatly to be feared, for at most, in such cases, a mere painful dryness of the throat results.

In children, however, the most careful instillation of even one or two drops may, by mere resorption into the conjunctival sac and the lacrymal ducts, produce some symptoms of poisoning, notably great congestion of the face, which is sometimes followed by elevation of temperature. Finally, certain adults are so susceptible to the action of this drug that the instillation of two or, at most, three drops is sufficient to produce very evident symptoms of general intoxication. In such cases *scopolamine* may be employed. Its mydriatic effect is very pronounced, and proves almost as lasting, on the whole, as that of atropine.

Atropine is uncalled for and fails to afford any marked relief to any pain which is caused by a superficial form of keratitis, and which is local and unassociated with ciliary neuralgia. In such cases the drug needlessly augments the photophobia, allowing more light to enter the eye. It is contraindicated when dilatation of the pupil is impossible and in cases in which there are severe hypopyon and iritis, or in which firm, old, anterior or posterior synechia are present. In such instances it produces an increase of ciliary pain and gives rise to a tendency for the eyeball to became hard. In deep ulcers it augments the danger of perforation by increasing the pressure in the anterior chamber and by paralyzing the intra-ocular muscles. (See article on *Mydriatics.*) If, then, in such cases, the action of atropine is not sufficient to dilate the pupil, it is better to abandon its employment and even replace it by the use of eserine. Peripheral prolapses of the iris are aggravated by the employment of atropine. Homatropine has a very feeble and quite transient action in corneal inflammation.

5. *Miotics.*—In keratitis, miotics diminish the tension of the eye, thus possessing an opposite effect to that which is observed from the employment of mydriatics. By their use the intra-ocular muscles are contracted and the irido-corneal angle is freed. (See article on *Miotics.*) Eserine may often be employed with good effect in ulcerations or in softenings of the cornea and for the prevention of threatened ectasis or perforation. It is also used for the purpose of reducing any peripheral prolapse of the iris. It can be employed in those cases in which it is desired to overcome any increase in the ocular tension that may have been caused by atropine. Its usefulness in certain forms of keratitis has led to its more or less general employment in all varieties of corneal affection, thus exceeding its scope, which should be to employ it only in well selected cases. Eserine certainly possesses no real antiseptic properties. One or more drops of a one or two per cent. strength solution of the sulphate, combined with corrosive sublimate, may be applied daily. Pilocarpine has been employed for the same purposes as eserine; it is probably less painful, but acts less energetically.

6. *Cocaine.*—This drug, which was brought forward by Koller, if used in the form of an aqueous solution in the strengths of four to five per cent., quiets for some fifteen minutes to half an hour the so-called "superficial pains," and renders the cornea insensible to painful applications, such as caused by the employment of the thermo-cautery and the crayon of methyl-blue. Its utility is incontestable in pronounced cases of irritable superficial keratitis, but it can be scarcely considered adequate as an exclusive treatment in this condition, while its abuse may lead to exfoliation of the epithelium and insensibility of the cornea, in themselves a cause of keratitis. Poisoning from its use is not to be feared.

7. *Narcotics.*—With the exception of hypodermatic injections of morphine, narcotics have but little effect in relieving the pain of keratitis. The remedy for the superficial form of pain is cocaine, while that for the ciliary variety is atropine. In cases in which the latter drug does not have the desired effect, a sedative action may be frequently obtained from the internal administration of antipyrin or phenacetin, in doses of twenty-five to fifty centigrammes, given in the form of powder, which may have to be repeated once or twice at half-hour intervals.

8. *Dark Glasses and Shades.*—The eyes of patients suffering from photophobia and blepharospasm should be more or less protected from light by means of card board shades and spectacles with very large (shell-shaped) glasses of some dark color. Smoked glasses are to be preferred to blue ones, as the blue rays irritate the retina. The selection of such glasses should not be left with the patient, as he is likely to secure too light a tint. By wearing such coverings to the eyes pain is lessened and the patient is able to go out of doors for the purpose of exercise.

9. *Revulsives and Local Abstraction of Blood.*—Revulsives and living or artificial leeches applied to the temple are of but little use in keratitis unless the condition be complicated by an irido-cyclitis.

## VARIETIES OF KERATITIS.

A rational division of the different forms of keratitis is impossible in the present state of knowledge, especially as there are not any means of subdividing them according to a fixed basis,—such as, for example, an anatomical one.

Nevertheless, a fundamental division of the different forms of keratitis, which is useful from a clinical point of view, is that into a superficial and a deep variety. The clinical characteristics are different in each, necessitating appropriate methods of treatment. These differences are based largely upon anatomical grounds. The anterior corneal planes (the conjunctival portion of the cornea) receive their nutrition in almost the same manner as the bulbar conjunctiva, while the deep planes have a nutriment supply that is more closely allied to that of the sclerotic, the iris, and the ciliary body. The anterior corneal planes are chiefly nourished by an anastomosis of the conjunctival vessels with those of the superficial ciliary vessels, while the deep ones receive their nutriment from the deep ciliary vessels. The anterior planes are enervated upon the periphery of the cornea by superficial nerves, while elsewhere they are supplied by deep posterior ciliary branches that arise from the

ciliary body. From an embryological point of view, the superficial corneal planes belong to the conjunctiva; the middle are derived from the mesoderm,—a part of the sclerotic, and the deeper planes appear to be reflected from the primary optic vesicle, just as in the case of the chorioid, the ciliary body, and the iris.

Certain disorders are to be found indifferently in the limbus conjunctivæ and in the anterior corneal planes (such as phlyctenular and superficial punctate keratitis). Most of the conjunctival maladies which invade the cornea primarily produce a superficial form of keratitis (such as granular pannus). The superficial varieties of inflammation of the cornea, rather than the deep ones, are accompanied by a congestion of the conjunctival vessels, while deep disorders involving the cornea, the iris, and the ciliary body bear an important relation to one another. In the superficial forms of inflammation of the cornea the newly created vascular channels originate from the superficial pericorneal vessels, while in deep keratitis the blood-supply is renewed from vessels that anastomose with those of the deeper trunks. The development of the deep primary disorders, which are often, if not always, of constitutional origin, as from syphilis, rheumatism, etc., takes place with extreme slowness. On the other hand, the superficial forms of inflammation are of comparatively rapid formation, and are rather due to traumatic causes or to ectogenous invasion by bacteria.

Another division of keratitis is into a bacterial and a non-bacterial form, in which suppuration, corneal infiltration, and ulceration are to be considered.

The variety of the lesion is sometimes sufficient to justify a distinction. Again, cases resembling each other clinically may exhibit an etiological difference.

The study of the varieties of keratitis may be arranged under the following heads:

A. Superficial keratitis, which is further subdivided in accordance with its etiology, the form of the lesion, source, etc.

B. Deep corneal inflammations that are (*a*) due to bacteria and suppurative; (*b*) non-suppurative and non-ulcerous, which consist essentially of infiltrations.

C. Cicatricial sequelæ of keratitis.

D. Corneal tumors.

These subdivisions are defective in more than one respect. A superficial keratitis may start as such, but later, owing to its extension, it may be necessary to class it with the deep variety; or the inflammation may be deep, and yet, owing to certain analogies as to its origin, will have to be placed with the superficial varieties. A keratitis by becoming infected may totally change its clinical course.

## A.—SUPERFICIAL KERATITIS.

In this variety of keratitis the anterior corneal planes, the membrane of Bowman, and often the adjacent corneal lamellæ are involved. An infiltration, which is sometimes vesicular, usually gives rise to a deposit that projects beyond the surface and which in turn is followed by ulceration and loss of substance. It is possible, however, for the erosion to be primary. Later, the new formation of corneal blood-vessels may play an important *rôle* in the development of pannus.—The general symptoms of keratitis consist in disturbances of the cornea, roughening of the corneal surface, absence of lustre, superficial ulcers, and ciliary injection. The seat

and extent of the lesion producing a general or partial ciliary injection is generally associated with a slight conjunctival injection. When blood-vessels are formed within the cornea, under Bowman's membrane, the epithelium is, as a rule, elevated by them, and they can be traced as far as the superficial ciliary vessels. Pain may be absent or be only of a minor character. The occurrence of ciliary neuralgia and contraction of the pupil should always awaken the suspicion that the affection has gained in depth and has become infected. The degree of unevenness of the surface naturally varies from roughness that is barely perceptible to one in which the elevations and depressions are plainly to be seen. A deep infiltration is one which is more apt to be diffuse, not so raised above the surface, and more tardy in becoming ulcerated.

Prognosis, as a rule, is favorable unless a severe secondary infection—which is always to be dreaded—supervenes. The amount of visual disturbance depends on the location of the trouble in relation to the pupil, and on its extent and character. The usual treatment is that which is indicated for superficial traumatic keratitis.

### A.—SUPERFICIAL TRAUMATIC KERATITIS.

*Traumatic injuries* to the cornea may be caused in the most diverse ways (see *Corneal Ulcers*); they may arise from external conditions, whether produced by the person himself or by other agencies, or they may originate within the protecting structures of the eye. A simple wound of the cornea usually heals in such a manner that it does not deserve to be called a keratitis. To constitute keratitis in a clinical sense the injury must be followed by microbic infection; mere repetition of the traumatism is rarely sufficient for this purpose. In such cases the intervention of microbes plays a leading *rôle.*

As external causes for traumatism solid particles floating in the air, rain, snow, wind, rubbing with the hand, detached bodies from larger objects, etc., may be cited. Disappearance of the eye-lashes naturally permits such objects more readily to gain access to the cornea. Within the structures normally used for protecting the eyes from injury, swellings and cicatrices of the conjunctiva, various conditions of the lids, deviated cilia, and crusts on the palpebral borders may be found. The lids may be deformed by congenital conditions, by traumatism, or by various affections, such as blepharitis and cicatrices of the neighboring skin, and in consequence no longer adequately protect the eye from agencies that ordinarily would not prove detrimental to the cornea. Again, the eye so affected remains partly open during sleep, and thus causes corneal desiccation and exfoliation. Anæsthetics, whether local or general, and paralysis of the trigeminus act in the same way. Diseases which lower general sensibility, as typhoid fever, must be included in the description of this condition.

Slight *wounds may become infected* through agencies of the most varied character; indeed, certain causes of infection operate so frequently that

they seem to induce keratitis alone without traumatism. Causes of infection are blepharitis, affections of the lacrymal passages or of the mucous membrane of the nose (ozæna), facial eruptions, etc.; more often than is generally supposed the infective germs are conveyed by the hands or on a handkerchief.

The symptoms observed are, generally speaking, those of the superficial forms of keratitis (see *supra*) and especially ulcers. Corneal vascularization rarely produces pannus, nor are well-marked maculæ to be feared unless the inflammation continues for a long period of time.

*Complications.*—Superficial forms of inflammation frequently give rise to the production of the deep variety of keratitis, this being especially true if the infection is of a virulent character.

*Treatment.*—The proper treatment consists in the removal of the exciting cause of the injury, if this be possible, and of the use of antiseptic lotions to the conjunctiva and the tarsal margins, for it is in these situations that bacteria find a favorable *nidus* for their multiplication. The application of a bandage is here of importance. Should affections of the lacrymal passages, blepharitis, cutaneous eruptions, or ozæna exist, they should receive appropriate treatment Yellow oxide of mercury ointment and other remedial agents employed in vascularized phlyctenules are of value in stimulating rebellious ulcers in which cicatrization and a filling of the excavation consist only in the corneal facet being covered with epithelium which gives rise to irregular reflections. Cure is often hastened by the scraping of affected areas with a curette. Peritomy may be employed in those cases in which an excessive vascularization does not yield to other methods of treatment. (See *Granular Pannus.*)

### B.—PHLYCTENULAR KERATITIS.

Phlyctenular keratitis (phlyctenular, lymphatic, or scrofulous conjunctivitis) is a disease affecting the corneal and pericorneal conjunctivæ of *infants and adolescents.* While the cornea and limbus conjunctivæ are the most frequent localities in which it is seen, it is sometimes, nevertheless, described among the conjunctival diseases under the name of pustular conjunctivitis, for the reason that the lesion is often situated on the bulbar conjunctiva. Conjunctival pustules may precede, accompany, or follow corneal phlyctenules.

Small opacities and protuberant infiltrations, that are improperly called phlyctenules, are found to occur, isolated or multiple, on the cornea and limbus conjunctivæ. Ordinarily, the infiltrations ulcerate and cicatrize, but successive eruptions of phlyctenules may be accompanied by such persistent vascularization that the malady lasts for a much longer time; such vascular forms when pronounced constitute the so-called scrofulous pannus. The appearance of these eruptions is accompanied by ciliary and conjunctival injection, and is followed by photophobia, lacrymation without mucus, and blepharospasm.

*Avascular Forms.*—(*a*) In this variety a single phlyctenule, which is usually of a small size and is often discovered with difficulty after ulceration has taken place, appears at or near the centre of the cornea. This is surrounded by simple ciliary injection. There is a slight degree of contraction of the pupil, and frequently intense photophobia and blepharospasm are found. (*b*) Here one or several phlyctenules, that frequently show vascularization after ulceration, appear on the periphery of the cornea. Ciliary and conjunctival injection is very marked at the level of the ulcerations, and, if partial, appears in the form of a triangle with its apex turned towards the phlyctenule of the cornea. (*c*) In this form the phlyctenules are found in the limbus conjunctivæ. They may be discrete, or exhibit themselves in the form of strings, causing the conjunctiva to swell and to constitute a pericorneal cushion for the ulcer. As a rule, the conjunctival injection is so intense that it masks the ciliary injection. (*d*) Although not avascular, the conjunctival phlyctenules, or pustulous conjunctivitis, may be included here. One or at most two infiltrations appear within a radius of half a centimetre around the periphery of the cornea. In this subdivision the size of the phlyctenule is larger, this being so because it occurs in a tissue of looser texture.

In the same subject may be seen successively or simultaneously the different manifestations of the various avascular forms of superficial keratitis just described.

*Vascular Forms.*—Here the peripheral phlyctenules are especially prone to undergo vascular changes. The newly formed vessels are superficial, and are situated close to Bowman's membrane, though they are never found situated under the epithelium. The corneal surface is elevated by the inflammatory vascular channels, the course of which can be traced to their origin from the ciliary vessels. The appearance of such vessels in the superficial forms of inflammation of the cornea causes the disorder to last longer than it otherwise would, whereas when the vessels are found in deep ulcerations their presence is favorable to the repair of the parts that are affected.

Two vascular forms are to be distinguished. (*a*) In *phlyctenular pannus* the phlyctenules may be united by the formation of new blood-vessels. They ulcerate, but show no disposition to heal. Exfoliation of the epithelium takes place on the edge of the persistent vessels. This process is followed by the appearance of a new crop of phlyctenules which undergo similar changes. In this manner segments of the cornea or even the entire membrane may become infiltrated, causing the surface to become bosselated and traversed by numerous blood-vessels. This variety of pannus scarcely ever attains such a degree of development as is observed in extreme examples of the granular form. (*b*) In *band-shaped keratitis* vascularization of a relatively large peripheral phlyctenule, which extends little by little towards the centre of the cornea, takes place. Behind the phlyctenule are to be seen strings of blood-vessels which run almost parallel to one another to the margin of the ulcer. These vessels have the appearance of

pushing the ulcer in the direction of the corneal centre or of being dragged by it.

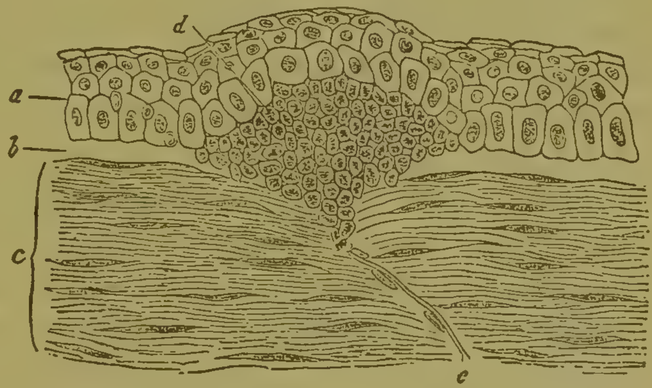
*Pathological Anatomy.*—The term phlyctenule, which denotes a blister, is badly chosen, as the vesicles rarely contain liquid, and when they do, the contents are generally turbid. These cellular infiltrations are situated in the superficial layers of the true corneal tissue, elevating and perforating Bowman's membrane and its epithelium. Even phlyctenules of small size, consisting of subepithelial cellular infiltrations, reach the outer layers of the cornea by nerve-channels (Ivanoff). Other authors could not find any relation between the infiltration and the nerve-canals. (Fig. 7.) While the infiltration may be very deep at the onset, the invasion of the deeper corneal planes is more often the result of a late intense infection by bacteria.

The newly formed blood-vessels are found to be situated beneath Bowman's membrane.

The anatomical details of band-shaped keratitis are but imperfectly understood.

*Etiology.*—Lymphatism and scrofula are predisposing causes for the production of phlyctenules, which, as a rule, appear simultaneously in both eyes. These conditions may not be manifest at first, but the long duration of the disease gives rise to anæmia and lymphatism. The coryza and the swelling of the upper lip, which are often cited as proofs of a lymphatic state, may be directly consequent upon the ocular disorder, without which they would not occur. When once established the phlyctenular disease is kept up by the coryza, the septic secretion being conveyed to the eyes by the hands or handkerchiefs. In some exceedingly rebellious cases adenoid vegetations may be found in the throat, and their extirpation is followed by rapid healing of the eyes. The enlargement of the submaxillary glands is frequently a consequence of facial eczema, which may have resulted from irritation of the skin by the secretions arising from the affected eye. On the other hand, the eczema may have appeared first, or have become prominent simultaneously with the outbreak of the phlyctenules, in which case they may be looked upon as eczematous in type.

Fig. 7.




Subepithelial cellular infiltration in a vesicle of phlyctenular keratitis (after Ivanoff).

*a*, epithelium; *b*, Bowman's membrane; *c*, corneal tissue; *d*, subepithelial cellular infiltration; *e*, nerve-canal, through which the migratory cells have reached the epithelium.

As has already been stated, phlyctenules are frequently situated on the limbus conjunctivæ; they are accompanied by intense conjunctival hyperæmia and a secretion of mucus, the so-called eczematous conjunctivitis of

Horner. At other times the conjunctival inflammation is secondary to the phlyctenules and is the result of their improper treatment.

This form of conjunctivitis is the most common ocular disease of childhood, and is essentially a malady of the early years of life. It is rarely found before the first year, appearing most frequently from the fifth to the twelfth. The condition is rarely seen after the age of twenty-five years. It must be stated that in children injuries or other causes of keratitis have a strong tendency to appear under the form of lymphatic cellular infiltrations.

The ordinary microbes of suppuration are found in the affected areas (Gifford), and, on the authority of Bach, staphylococci are always to be obtained from the small areas, provided that the examination be made on the first or the second day. As this same variety of organism is found also in eczematous eruptions, the relationship which is said by Horner to exist between the phlyctenules and eczema would be thereby confirmed.

*Course and Symptoms.*—There is no disease of the eyes in which photophobia and blepharospasm play a more important part than they do in phlyctenules. This seems to be due to the numerous superficial nervous filaments becoming distended and compressed, either in the epithelium covering the cornea or in their passage through the membrane of Bowman.—An eruption of phlyctenules may heal with considerable rapidity. Ordinarily, however, there is a marked tendency for relapses to occur, fresh eruptions following one another in rapid succession or appearing after the lapse of variable periods of time. In obstinate cases, it is a pitiable sight to see the affected children shrinking for weeks and months from daylight by various devices, such as seeking dark corners and burying their heads in cushions. The lack of air and of light lowers the general nutrition, and produces anæmia and lymphatism, if these conditions do not pre-exist. The tears, which normally possess an alkaline reaction, are often mingled with the irritating secretions from the eye, and thus produce a conjunctivitis which is sometimes purulent. Excoriations of the skin of the lower lids, fissures at the external palpebral angles, coryza, and swelling of the upper lip may occur. Persistent blepharospasm leads to an œdematous swelling of the upper lid, which later may extend to the lower one. The brows are contracted by the spasm of the lids, and the eyelids are closed for the purpose of shielding the eyes as much as possible from the light.

*Complications.*—When the infection is intense, it may produce suppuration; or a small corneal phlyctenule, which can be discovered only with difficulty, owing to the intense involvement of the cornea, may appear as the point of entrance for the production of a profuse deep keratitis (parenchymatous keratitis). Such a condition is made manifest by intense ciliary pain, contraction of the pupil, and an intolerance to the employment of ordinary medications. Perforation is often produced by peripheral phlyctenules. Conjunctival catarrh may be simulated by a copious secretion from the conjunctiva. It is well to remember in this connection that simple

(non-follicular) catarrh is nearly unknown from the fifth to the twelfth year, the time at which this affection is most likely to be observed. Blepharitis, which is likewise an expression of lymphatism, and usually, if not always, due to staphylococci, is often observed as a concomitant or a cause of phlyctenules.

*Prognosis.*—This is favorable, unless infectious complications supervene, the latter too often being the result of negligence. Minute ulcers heal without leaving an appreciable macula. Dense, permanent maculæ are ordinarily the result of large phlyctenules, especially if they have assumed the vascular variety. The track of band-shaped keratitis can always be traced by the resultant opacities.

*Treatment.*—As this condition is often due to a general cachexia as well as to local causes, treatment must usually be directed to both of these conditions.—Local measures alone are sometimes efficacious, and should vary according to the clinical forms which are assumed and the complications that are present. Finely powdered calomel applied once daily with a dry hair-pencil is a sovereign remedy in simple non-vascular phlyctenules that are unaccompanied by conjunctival secretion. The chemical composition of the mercurous chloride (calomel) is gradually converted by contact with the secretions of the eye into mercuric chloride (corrosive sublimate). Care must be exercised not to use too great a quantity of calomel, as it accumulates in the inferior *cul-de-sac* and acts as a caustic on the conjunctiva. The simultaneous use of the iodides internally and calomel locally is contraindicated, as the former drug appears in the lacrymal secretions and converts the calomel into the iodide of mercury, which acts as an irritant.—The mild chloride of mercury is of no value in the treatment of phlyctenular pannus, and is especially useless in band-shaped keratitis. Here the ointment of the yellow oxide of mercury exerts a most beneficial action, particularly if the conjunctival secretion is not abundant. A small amount of the ointment, prepared in the proportion of one-half to one centigramme or more of the yellow oxide of mercury to one gramme of vaseline, should be placed daily on the edges of the eyelids and distributed over the eyeball by gentle massage made through the upper lid.—The applications of calomel and of the yellow oxide of mercury ointment should be made by the physician himself. For continuous use lotions of the bichloride of mercury may be prescribed. Conjunctival secretions and eczematous forms of the disease should be treated with weak astringents, such as boric acid lotions, and antiseptics, such as solutions of corrosive sublimate. After the secretion has been diminished by these methods recourse may be had to the use of calomel and the yellow oxide of mercury. —Atropine is without effect in lessening photophobia and blepharospasm. The latter condition may be made to disappear temporarily by the production of slight asphyxiation. This barbarous method of relief is secured by holding the child's nose, the face being plunged for a brief period of time in water.—The wearing of dark glasses and of a visor are often

helpful. Prolonged application of cocaine is sometimes of value.—Experience has shown that the wearing of a bandage is not only useless, but that it often acts unfavorably, even in cases in which the secretions are not copious. It may, however, be of value in those cases in which it is desired to prevent infection of the eye from excoriations of the skin and coryza.—Facial eczema, nasal catarrh, and any cause of special infection demand appropriate treatment. For the eczema the ointment of the oxide of zinc may be employed. The coryza may be lessened by the use of sufficiently strong ointments of the oxide of mercury, or by insufflations of powders containing equal parts of bismuth, boracic acid, and camphor. In this condition astringent injections are of use. If in rebellious cases adenoid vegetations of the pharynx be found, their extirpation is the necessary and very efficacious treatment. The fissures of the external angles may be touched by a crayon of the nitrate of silver.—For simple phlyctænulæ daily instillations of eserine have been recommended by certain authors. The drug, however, is badly tolerated, and its efficacy is doubtful.—In band-shaped keratitis cauterization of the ulcer by heat is ordinarily superfluous. If the ointment of the yellow oxide of mercury does not suffice in effecting a cure, curetting of the ulcer by means of a Daviel spoon appears often to be valuable in shortening the duration of the affection (de Wecker). Peritomy is superfluous, unless it is employed in the rebellious forms.—It is important to remember that the use of revulsives in the treatment of the disease should not be permitted.—The severe infection of a phlyctenular eruption, with penetration to a great depth, contraindicates the employment of the calomel and of the yellow oxide of mercury ointment. On the contrary, the condition calls for a treatment that is applicable to deep ulcers,—namely, bandages, antiseptic lotions, atropine, etc.

General treatment is important. It should consist in good nourishment, cod-liver oil (if there be scrofula), quiet, fresh air, frictions of the skin, and salt baths.

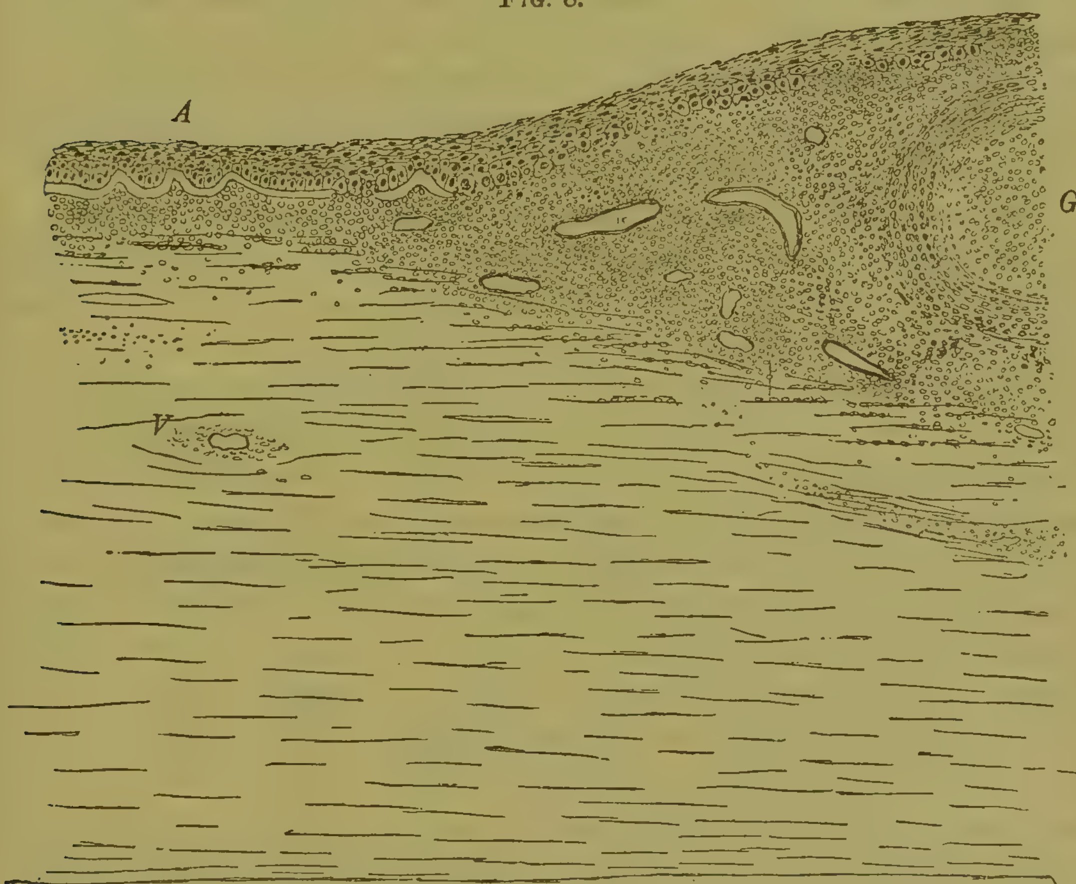
As the conjunctival sac continues to be infected with pathogenic microbes even when the phlyctenular eruption has disappeared, just as the edges of the eyelids remain infected for a long period of time after styes, the necessity of continuing antiseptic treatment, especially the use of sublimated lotions, for a considerable period of time (several weeks) is important. Relapses are always to be feared, especially in the non-vascular forms.

### C.—GRANULAR PANNUS.

In cases of conjunctival granulations a superficial keratitis, which has a strong tendency to become vascularized, is almost always produced. Starting from the superior corneal margins, this condition exhibits a slight epithelial exfoliation with moderate infiltration and the appearance of numerous vascular branches that are derived from the superficial pericorneal vessels. Marked projections are occasionally produced by the con-

junctival vessels penetrating the diseased portions. As a rule, this form of keratitis may involve the superior half, or even the whole, of the cornea. The disturbances of the cornea and the inequalities of its surface afford a favorable opportunity for multiplication and enlargement of the vessels. In extreme cases—that is, in *pannus crassus* rather than in *pannus tenuis*—the corneal surface is bosselated and fleshy, suggesting the appearance of a granulating wound.—The aspect first presented by this form of pannus is that of an epithelial exfoliation which has resulted from the friction of a bosselated cicatrized conjunctiva, or from deviated cilia. Vascular infiltrations of the anterior corneal planes, beneath Bowman's membrane, which still remains intact, are seen. (Fig. 8, *A*.) This membrane is finally perforated, and the epithelium is raised by the growth of the new vascularized

FIG. 8.




Granular pannus of the cornea. At *A* the cellular infiltration is seen under the wrinkled plicated membrane of Bowman. Towards the right it has attacked and pierced this membrane, which has disappeared to a large extent. At *G* a portion of a follicle which is surrounded by a membrane-like structure and by a real trachomatous granulation, included in the pannus tissue, can be seen. *V* represents a vessel that is surrounded by perivascular infiltration. The lower part of the epithelial lining, where it touches the granular tissue, is badly outlined. The cellular infiltration should penetrate it more or less.

tissue that is situated beneath it. In pannus crassus (Fig. 8, *G*) non-vascular follicles are sometimes found in the midst of the granular tissue. These are true follicles, surrounded by a membrane-like substance.

*Diagnosis.*—Granular pannus may be confounded with phlyctenular pannus (*vide supra*) and with certain forms of superficial (traumatic) vas-

cular keratitis. It is only in exceptional cases that granular pannus starts not from the superior part of the cornea, and the examination of the palpebral conjunctiva will remove all doubt as to whether or not this is the case.

*Prognosis and Sequelæ.*—Granular pannus is characterized by its extreme persistence. If imperfectly treated, pannus crassus may last for years, and by its presence the sight of the affected eye may be destroyed. Pannus tenuis may also produce permanent maculæ that disturb vision more greatly when they are situated in front of the pupil. The cornea, weakened by the cellular infiltration of the deeper structures, may yield to the intra-ocular pressure, the central curvature usually increasing (keratectasis). Finally, pyogenic microbes may invade the cornea, and thus give rise to suppuration, which takes place especially in the centre of the cornea, and may lead to perforation. Almost without exception, this process is followed by the appearance of anterior synechiæ and of corneal staphylomata which are due to softening of the corneal membrane. The suppurative processes may also invade the deeper structures of the eye and give rise to panophthalmitis.

*Treatment.*—Mild cases of pannus are to be treated with irritants in the same manner as granular conjunctivitis. Ordinarily, such remedial agents influence favorably the course of the disease, preventing the necessity of other special measures. When the keratitis tends to become deep or infected, irritating medication is to be replaced by the use of antiseptic lotions and atropine. Should irritant drugs give rise to persistent ciliary pain and augment the excitability of the eye, they should also be discontinued. In such cases the writer has recently found that instillations of a two to five per cent. strength solution of protargol are peculiarly efficacious. If the secretions be not too abundant, an occlusive bandage may be applied concurrently with the application of iodoform. In some instances recourse may be had to eserine, and iridectomy may be necessary to avoid an ectasis.

*Jequirity.*—The pannus sometimes disappears after suppuration resulting from a blennorrhœic or gonorrhœic inoculation. This heroic remedy was much used about the middle of the present century. In very intense double pannus crassus, surprisingly good results may be obtained from this form of treatment. It must, however, be borne in mind that a portion of the cornea which is not covered by the pannus may perforate, thus probably leading to a secondary affection of the fellow-eye. Fortunately, we now have in the seeds of jequirity a vegetable irritant which is more easily regulated and less repulsive than the blennorrhœic material formerly used (de Wecker). The seeds contain an amorphous substance known as jequiritine, the chemical composition of which is badly defined. When the seeds are placed in water (Venneman and Bruylant, Salomonsen, Sattler), this substance appears to develop by sprouting. It provokes a condition in the conjunctiva resembling that which is seen in purulent conjunctivitis that is due to the presence of bacteria. According to the latest formula, given by

de Wecker, three to five grammes of the seeds freed from their cortex are powdered and macerated for three hours in one hundred grammes of water, which should be used cold, as heat destroys the jequiritine. The freshly prepared filtered liquid is applied over the conjunctiva of the everted upper lid, and even placed over the cornea in very intense pannus. This is done with a tampon of cotton. After one or two days a single application gives rise to a conjunctivitis, with croupous plaques resembling those that are seen in cases of mild blennorrhœa. The conjunctivitis so induced is intensified by repeated applications or may be maintained at will. If the conditions are favorable, the pannus disappears.

In the application of this remedy the following indications for treatment should be present, otherwise the pannus may not heal and great danger to the cornea may be incurred:

1. The pannus should be complete; a non-vascularized portion of the cornea threatens to perforate.

2. The cornea of the second eye should be more or less vascularized throughout, the conjunctivitis being of a contagious character.

3. The palpebral conjunctiva should be the seat of infiltrated granulations or be more or less cicatrized. When the amount of secretion is considerable, the jequirity should not be employed, as it excites too violent a reaction.

Under the conditions just mentioned, jequirity is in a manner a specific for granular pannus, but it is not suitable, as a rule, for the treatment of general granular conjunctivitis.

Certain authors favor peritomy in the treatment of rebellious forms of granular pannus. Its use is even more general in obstinate cases of superficial vascular keratitis. Some recommend that simple incisions down to the sclerotic be made repeatedly, even advising that this should be done daily. This is done with a view of bringing the ciliary vessels into use during the time of repair. Others excise the conjunctiva around the cornea to the extent of half a centimetre's distance, and even cauterize the bottom of the wound for the purpose of destroying the ciliary vessels. These are very painful methods of treatment, even though cocaine be used in their performance. The writer has never employed any other method than that of oft-repeated incisions.

Eyes are often met with which have been cured of their granulations, but in which the conjunctiva is covered with cicatrices that look as though they were embossed. The rubbing of the eyelids produces incessant and ever-recurring exfoliations of the cornea. These are cases of traumatic keratitis which must be treated as such, special care being taken to avoid stimulants. Daily massage of the eyeball through the upper lid and the introduction of the ointment of the yellow oxide of mercury into the conjunctival sac are often the best means of treating this condition.

### D.—ACNE OF THE CORNEA; CORNEAL HERPES; ZONULAR HERPES; DENDRITIC KERATITIS; RODENT ULCER; KERATALGIA.

1. *Acne of the Cornea.*—Acne of the face, nose, or cheeks may be accompanied by eruptions on the cornea which have the appearance of small phlyctænulæ. These eruptions of acne of the cornea are found only in persons who have passed the period of adolescence. They are accompanied by marked redness of the edges of the eyelids, are extremely rebellious to treatment, and are prone to relapse. Calomel, antiseptic lotions, and the yellow oxide of mercury ointment have but little beneficial effect upon them.

2. *Febrile Herpes* (Horner).—An eruption of the transparent vesicles frequently appears in febrile or catarrhal maladies, especially in disorders of the respiratory tract, such as bronchitis, pneumonia, and influenza. The outbreak is often seen concurrently with herpetic efflorescence on the edges of the nose, and is accompanied by pain, lacrymation, etc. The vesicles are more or less numerous, are often arranged in groups, and are not larger than the head of a pin. The walls of the vesicles are composed of epithelium and some lamellæ of corneal tissue, and enclose a transparent liquid. When they burst, the resulting ulcer is slightly infiltrated. In the course of three or four weeks' time the wound heals without leaving visible traces of its former presence. Relapses are greatly to be feared. If the condition be neglected, the ulcerated area may become infected, infiltrated, and undergo suppuration. Sometimes these vesicles are from the first deeper and more infiltrated, this stage being the transition form to that of *dendritic keratitis.*—Under the name of *idiopathic herpes* has been described a variety of eruption of corneal vesicles that is analogous to those of febrile herpes, but which is not complicated either by facial herpes or by fever. Cases of this kind are usually classified under the term of filamentous keratitis; in part they are epithelial vesicles, which will be considered under the heads of *Vesicular Keratitis* and *Bullous Keratitis.*

*Treatment.*—This consists of antiseptic lotions, a bandage, and atropine, if necessary.

3. *Zonular* (*Ophthalmic*) *Herpes* (see also special article on *Herpes*).—In neuralgia of the trigeminus one, two, or three foci of intense infiltration sometimes unexpectedly appear, often elevating the surface of the cornea, and always reaching a certain depth. Neuralgia of the ophthalmic branch, and especially of its nasal subdivision, has this effect, whether it be or be not accompanied by an eruption of herpetic pimples, herpes of the skin, of the forehead, of the cheek, or of the nose, and by anæsthesia in the region of the eye. Now and then the affection begins with a group of small protuberant infiltrations, which resemble an eruption of febrile herpes, and soon become confluent. Generally, the symptoms are more severe, and are accompanied by congestion of the iris or even by an iritis. The infiltrations develop into ulcers which tend to produce perforation and hypopyon.

Healing takes place but slowly. Relapses are greatly to be feared as long as the neuralgia and insensibility of the cornea remain. The eruptions appear to have the same significance as those of zonular herpes of the surrounding skin, with which they are often associated,—*i.e.*, they result from a peripheral neuritis.[1] This condition must not be confounded with the exfoliations that are so often observed in the inferior half of the cornea in cases of anæsthesia of the ophthalmic nerve, and which in their final development produce a suppurative neuroparalytic keratitis.

Zonular herpes of the cornea always reveals the characteristics of an infected and ulcerating deep corneal infiltration, and as such might have been discussed under *Deep Keratitis.*

*Treatment.*—This comprises dilatation of the pupil with atropine, the use of special antiseptic measures, such as corrosive sublimate lotions, and the application of the crayon of methyl-blue to the ulcer. Cauterization by heat may be recommended. The neuralgia should receive appropriate treatment.

4. *Dendritic Keratitis; Malarial Keratitis* (see special article).—This form of inflammation of the cornea (described by Hansen Grut, Emmert, Kipp, Noyes, Gillet de Grandmont, etc.) sometimes results from causes similar to those which produce febrile herpes of the cornea, and at other times the etiology is entirely different. The special form and course of the ulceration make it a separate clinical species. It begins by a circumscribed but rather intense infiltration which penetrates to a certain depth. The infiltration then ulcerates; the borders of the ulcer, may be perpendicular or more or less hollowed and infiltrated. New eruptions appear around the ulcer, which enlarges principally in one direction, and forms a furrow on the corneal surface. The affected area has at first the appearance of a comma, but as time goes on it elongates until, finally, it becomes arborescent and ramified. Photophobia, ciliary pain, congestion of the iris, and even iritis occur. When neglected, the ulceration may suppurate and perhaps perforate the cornea, though under proper care it has not this tendency. Its development resembles that of a suppurating, corroding (rodent) ulcer, or ulcus serpens, though its tendency to ulceration is less and the ulcer is more contracted. After healing all remaining opacities assume the arborescent form, which is also more or less the appearance of deep keratitis.

*Etiology.*—Febrile herpes may be transformed into dendritic keratitis. The writer has seen corneal phlyctænulæ assume this character. Kipp has often found this lesion associated with paludic poisoning, and calls it malarial keratitis. Emmert has isolated a microbe (a bacillus) which he is inclined to regard as the special pathogenic factor (mycotic keratitis); it has not been found by other observers, but the very peculiar progress of the ulcer seems to demonstrate that microbes play an important *rôle* in the production of the condition.

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[1] See special article on Eye Affections due to Graves's Disease and Herpes Zoster, in this System.

*Treatment.*—The treatment is the same as that for infected ulcers, including atropine, bandage, and antiseptics. The margin of the ulcer should be touched with a crayon of methyl-blue, and in obstinate cases it should be touched with a hot iron.

5. *Rodent Ulcer.*—Under this name, Mooren, Fuchs, and others have described a rare affection of the cornea, which is similar to the grave varieties of dendritic keratitis, and of which I lately observed an example in a young man. It has, however, been seen most frequently in old people. A burrowing, peripheral infiltration is transformed into an ulcer with thickened borders, which may become vascular and tend to cicatrize, when suddenly a neighboring portion of the cornea becomes infiltrated, ulcerated, etc. This process continues until the anterior layers (the epithelium and anterior lamellæ of the corneal tissue) become elevated through their entire extent. The ulcer extends in furrows, which are larger and deeper than those of dendritic keratitis. The centre of the cornea may be invaded, finally emerging as a protuberant and transparent islet. The entire cornea is at last covered by a very dense leucoma, and vision is almost abolished. Prognosis is indeed grave when both eyes are attacked, either simultaneously or successively ; for, as in the dendritic form of keratitis, the ulcer, being relatively deep, shows no marked tendency to suppurate, but only to destroy the superficial corneal lamellæ. Hypopyon is rare.

*Treatment.*—Only cauterization by heat (galvano-cautery) to the infiltrated border of the ulcer seems to be adequate to arrest the progress of this grave affection (Fuchs). Corrosive sublimate, pyoktanin, etc., are useful only as adjuvants. Ectasis of the shrunken cornea should be combated by the employment of the compress bandage and eserine.

### E.—FILAMENTARY KERATITIS.

In persons of advanced age the eye suddenly becomes irritated and weeps a little; there are slight pericorneal injection, pain, and photophobia. On the cornea appear what seem to be clear vesicles ; most of them are small, but the largest have a diameter of from one to one and a half millimetres. These apparent vesicles are tiny globules attached to a slender pedicle. The smallest are swept off by the lids, the larger ones are transformed into filaments, of a millimetre or less in thickness and half a centimetre or more in length, which hang flabbily over the front of the cornea. Winking raises the filaments, which, however, fall into place again. After some days or weeks there is a new eruption, which is followed by an exacerbation of the irritating symptoms. The affection may continue for months or even years. These eruptions sometimes consist of only one or two filaments; again there may suddenly appear a great number, a dozen or more, of all dimensions. The idiopathic variety of filamentary keratitis is, on the whole, a rare one, and has no tendency to become aggravated. Filaments similar to these may be present in chronic ulcers, their production ceasing as soon as healing of the lesion occurs.

*Anatomy* (Leber, Uhthoff, Fischer, Czermak, Nuel, and Hess).—In the midst of the epithelium of the cornea appear nests of clear, round cells, which are often polynuclear. The surrounding cells elongate, and force these nests in front of the epithelial plane. An envelope of flattened cells, that are undergoing mucous metamorphosis, and a pedicle made up of elongated cells are then formed. (See Fig. 9, *A*.)

Fig. 9.


Epithelial excrescence in filamentary keratitis. *A*, globular form; *B*, filamentous form of the excrescence (free extremity of the filament); *T*, central spiral portion of the pedicle; *N*, vestiges of hyaline cells driven out of the epithelium. These are in the form of a granular nucleus surrounded by a capsule of epithelial cells that have more or less undergone mucoid metamorphosis.

The pedicle is composed of epithelial fibres, which are elongated into fibrillæ, and rolled into spirals like a cable. This subsequently lengthens; the superficial fibrillæ, growing more than the others, emerge from the spiral and form for it a loose fibrillar envelope, completely impregnated with a mucoid substance. This constitutes the process during the course of the development of the filament. (Fig. 9, *B*.) The corneal tissue itself remains normal. The filaments are, therefore, formed entirely from the epithelium, and are similar to the horny excrescences of the epidermis. The hyaline cells (*N*), originally situated within the epithelium, bear some resemblance to those parasitic organisms known as coccidia, that are often seen in the centre of the epithelial nests of epitheliomata. The only wonder is that after the parasitic nests have been driven out of the corneal layer the repelling epithelial cells continue to grow and undergo mucous transformation.

*Diagnosis.*—Eruptions of filamentary keratitis have sometimes been confounded with herpes of the cornea. The initial globules and the short filaments have been supposed to be vesicles, while the long filaments have been taken for the shreds of ruptured vesicles.

*Treatment.*—The removal of the filaments gives immediate relief, but subsequent eruptions occur. The intra-epithelial nervous filaments are forced into the pedicle. The rather violent pains that accompany every new eruption are due to a pulling and tugging upon the nerves. The pain suddenly ceases on the removal of the filaments. The employment of a two per cent. aqueous solution of ammonium chloride appears to diminish the eruptions by favoring the exfoliation of the epithelium. Sattler has recommended shaving off the epithelium. In one of the writer's cases the filaments continued to be produced from time to time during several years, notwithstanding the employment of various treatments, such as the instillation of collyria of methyl-blue, as recommended by Sourdille.

Similar filaments are expectorated by asthmatics (Curschmann, Nuel). Certain cases of bronchial asthma are due to epithelial eruptions of this kind in the bronchial tubes.

### F.—SUPERFICIAL PUNCTATE KERATITIS.

Under this title is described (Fuchs, von Reuss, Adler, Groenouw) a superficial disease of the cornea, which by reason of its symptoms and anatomical lesions (Nuel) constitutes a morbid form that is entirely distinct from those which have already been described. In the past it has been confounded either with catarrhal conjunctivitis, *Schwellungskatarrh* of the German authors, or with a form of superficial keratitis which is not well defined. It is usually bilateral, both eyes being attacked simultaneously or after many days' interval.

*Symptoms.*—The disease begins with marked ciliary and conjunctival injection, slight secretion of mucus, pain, photophobia, and contraction of the pupil. This is the catarrhal stage, or stage of invasion. There next appear on the cornea small, superficial, yellowish-green spots that are fairly well outlined. They number from five or ten to fifty or more, the largest having a diameter of one millimetre, but most of them being smaller. In searching for them a magnifying glass should be used, otherwise the real nature of the affection may be overlooked. At the same time there is a diffuse œdema, also superficial, which has a smoky appearance, and is sometimes striated. The surface of the cornea is dull, especially on the level with the larger spots; and it may also be raised or studded with small knob-like projections. The eruption is often located in the centre of the cornea; at other times it may be situated nearer the periphery, in which case the limbus conjunctivæ is swollen, and there are small elevated spots that resemble phlyctænulæ, but which do not ulcerate. In the last case there is some secretion, but there is neither suppuration nor iritis. Some days after the eruption has appeared the symptoms suddenly cease and the œdema disappears. The corneal spots persist for weeks or even months, and go away by the process of resorption.

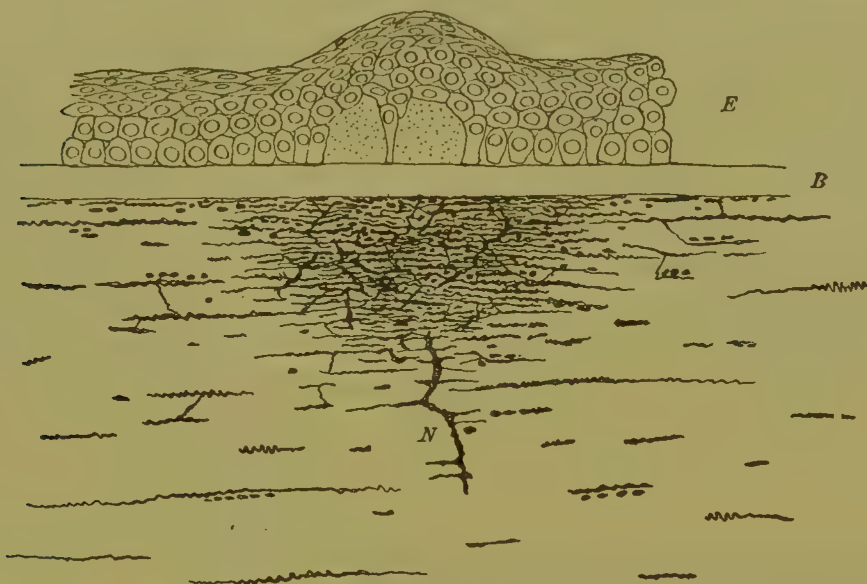
*Etiology.*—Superficial punctate keratitis is a disease of frequent occurrence between the ages of twenty and thirty-five years. The greater number of such patients have been exposed to intense cold. It is seen but little except in winter, and has been observed as a complication of influenza.

*Anatomy* (Nuel).—The diffuse corneal inflammation is produced chiefly by a marked œdema of the anterior corneal lamellæ. The spots are produced by filaments of fibrin undergoing hyaline change, which appear in contact with the fixed cells of the cornea. These filaments rolled in spiral form are situated between the corneal lamellæ; they are colored in intense red-violet by hæmatoxylin and stain deeply with aniline colors. (Fig. 10.) These filaments condense in places near the membrane of Bowman, and resemble thick felt. They constitute the spots described above. At the

level of these areas there is an œdema of the epithelium in the form of studs or knob-like epithelial projections that are transformed into epithelial lacunæ.

In a case of this character which recently came under the writer's observation, the nests of spiral filaments were overwhelmed by foci of micrococci that were lodged between the epithelial cells. In Fig. 10 the central

FIG. 10.




Longitudinal section of a macula in superficial punctate keratitis. *E*, epithelium of the cornea; *B*, Bowman's membrane; *N*, nerve-canal infiltrated with hyaline filaments arranged in coils. Under the macula in the centre of the cut are seen two lacunæ in the epithelium, elevating the latter.

deposits in the epithelium are the seat of the micrococcic invasion. The disease is, therefore, of bacterial origin, and the spiral fibres are probably products of bacteria, analogous to the spiraloid threads, often of large size, which have been described by Loeffler and others as present in growths of various other bacteria.

Superficial punctate keratitis is characterized by an inflammatory œdema of the anterior corneal planes, due to exogenous (epithelial) infection by a not well-identified coccus (staphylococcus, according to Valude). At no stage is there an infiltration of migratory cells. The lesions are located in the same places as phlyctænulæ.

*Treatment.*—All irritant medication should be abstained from. At the most, lotions of boric acid may be employed when there is secretion. Atropine may be used to counteract any contraction of the pupil. Salicylate of sodium internally has no effect. The exfoliation of the epithelium may demand the bandage. Dark glasses should be prescribed for the photophobia.

G.—VESICULAR AND BULLOUS KERATITIS; CALCAREOUS KERATITIS.

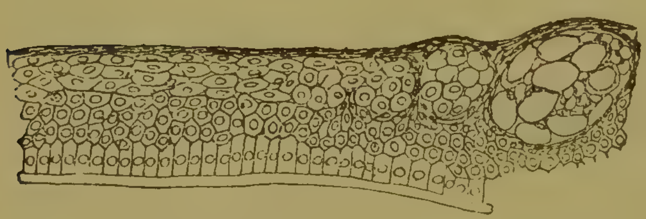
*Vesicular and Bullous Keratitis.*—These pathological forms appear to affect only the epithelium, or at most the superficial lamellæ of the cornea. They are found in eyes having large corneal cicatrices or staphylomata,

and which are often glaucomatous, blind, and lost in consequence of irido-cyclitis. Sometimes they appear in eyes which are not otherwise diseased. Accompanied by symptoms of rather violent irritation,—ciliary pain, ciliary injection, and lacrymation,—there appear suddenly one or more tiny vesicles (vesicular keratitis) or very large bullæ (bullous keratitis), with clear liquid contents, and with a covering which is composed of epithelial cells. In a few days' time the vesicles burst, cicatrization follows, and all symptoms of irritation disappear. Some weeks later fresh eruptions take place. Such eyes, besides being useless, are a permanent cause of suffering. Often painful and very rebellious epithelial ulcerations appear, known as the "relapsing ulcerations" of Arlt, and atheromatous ulcers. The nutrition and the sensibility in eyes of this class of cases are profoundly altered.

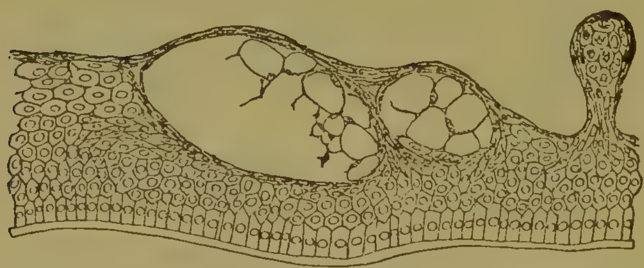
The vesicles appear to have a double origin: 1. The lymphatic stasis of glaucomatous eyes produces an interstitial œdema of the epithelium, which tends to the production of vesicles, etc. (Fuchs). 2. The continual irritation of the corneal epithelium alters the nutrition of the cells. Above all, the cells of the middle layer are transformed into clear vesicles (Fig. 11), which in places acquire a very large size. The cellular membranes of these large vesicles are destroyed, and in this manner a lacuna is produced, which is often rather large and becomes more and more distended; such a vesicle is covered by epithelium, that separates it also from Bowman's membrane (Nuel). The diffuse hyaline and mucous alterations of the epithelium also produce those epithelial ulcerations which are often so painful in eyes that are disorganized by glaucoma or otherwise.

Fig. 11.

*A*



*B*



*A*, hyaline degeneration of the corneal epithelium, capable of producing the formation of large epithelial vesicles (bullous keratitis); *B*, firm excrescences similar to those of filamentary keratitis.

The first mode of formation produces vesicles covered by epithelium and resting upon Bowman's membrane; the second method furnishes the intra-epithelial vesicles. Vesicles which lie in the substance of the corneal membrane have also been described; they are said to be developed underneath Bowman's membrane. This point, however, merits further research.

Each eruption of vesicles is accompanied by pain, which diminishes as the attack recedes. As in filamentary keratitis, the pain seems to be due to an irritation of the interepithelial nerve-fibrils.

*Treatment.*—The removal of the vesicles and of the bullæ gives relief.

Attempts to prevent their recurrence by eradicating the seat of the eruptions by cauterization have been unsuccessful. Such procedures will not cure either the lymphatic stagnation in glaucomatous eyes or the diffuse degeneration of the epithelium. The enucleation of such eyes, the sight of which is lost, is often obligatory. Iridectomy performed for the purpose of improving the nutrition of the cornea is of doubtful efficacy. Posterior sclerotomy is to be preferred to iridectomy.

*Idiopathic Vesicular or Traumatic Vesicular Keratitis.*—A vesicular keratitis in eyes which are otherwise normal has also been described; it occurs spontaneously, most frequently after slight traumatisms. According to all appearances, most, if not all, of these forms belong in the class of filamentary keratitis.

*Calcareous Keratitis; Ribbon-Shaped Corneal Opacity.*—This form of inflammation should be compared with bullous keratitis, accompanied as it also is by an alteration of the epithelium in disorganized eyes. In eyes that are lost through glaucoma a middle horizontal zone of the cornea is affected; it becomes dull and slightly raised. (See this pathological form discussed under *Consequences of Keratitis.*)

## B.—DEEP KERATITIS.

These inflammations will be here treated of under the two subheadings (*a*) deep ulcerative and suppurative keratitis and (*b*) deep non-suppurative keratitis. As already stated, those of the first subdivision are caused by exogenous microbic infection, and those of the second by endogenous, microbic or non-microbic infection. Cases of the first-class exhibit the characteristics of a local disease; those of the second are generally of constitutional origin.

### A.—DEEP ULCERATIVE AND SUPPURATIVE KERATITIS.

(*a*) **Ulcers of the Cornea.**—An ulcer of the cornea is practically a loss of substance which is or has been progressive,—that is, has invaded the surrounding tissues. Some ulcers result from injuries to the cornea, and others take their origin from cellular infiltration which has assumed an ulcerative form. In most cases of superficial keratitis ulceration may appear. No distinct line of demarcation can be drawn between the simple exfoliations which heal rapidly and the more serious ulcers. Corneal suppurations are reserved for a special chapter, although they merge insensibly into non-suppurative types. The principal features of all ulcers appear to be an infection by the pathogenic microbes, the yellow and white staphylococci, the pneumococcus, the streptococcus, and, exceptionally, bacilli that have not as yet been well defined. When the microbic infection is pronounced, there is suppuration. The greater frequency of corneal as compared with conjunctival ulcers is due to the fact that the cornea is not supplied with blood-vessels. Thus it follows that the nutrition of this membrane is less well assured, and the struggle against microbes is less efficient.

1. *Progressive Ulcers.*—Extension in depth and width of the ulcer is caused by the appearance of new infiltrations. The borders show an opaque, yellowish or whitish infiltration, which is sometimes uniform and sometimes appears as radiating bands. The intralamellar spaces may be infiltrated and hollowed out so as to constitute pockets that are filled with pus. The border of the ulcer is encircled by an œdematous areola which spreads widely over the cornea or merges insensibly into the surrounding tissue. The floor of the ulcer is often irregularly studded with infiltrated areas or necrosing corneal lamellæ, that are in the process of being cast off by the inflammatory process. Sometimes, however, the floor may be transparent and but slightly infiltrated, especially when it is composed of only the membrane of Descemet. The depth of the excavation does not generally correspond with the extent of the destruction of tissue; the lamellæ of the floor, being swollen, may even project above the surface. Extension may sometimes be greatest in depth (boring ulcer). At other times it may increase in size (rodent ulcer), and again enlarge in some particular direction. These are serpiginous ulcers, of which dendritic keratitis is an example. Another illustration is the ulcus serpens of Saemisch, for a description of which see *Suppurative Keratitis.* Summing up, the floor of the ulcer is irregular, spotted, and rarely transparent through its entire extent. The borders are almost always infiltrated. Over the floor and at the edge the infiltration is intense and of a yellowish-white color whenever the suppuration has a tendency to be profuse. The eye is irritated, and extreme ciliary injection, lacrymation, photophobia, and ciliary pain are present. The iris reacts but slowly to light, and its tissue is congested in every case in which the ulcer is at all deep. The iritis may be complicated with posterior synechia or hypopyon. (See *Suppurative Keratitis.*) The pain and photophobia may be absent even in very grave ulcers, when they have assumed a torpid character.

2. *Retrogression of Corneal Ulcers.*—When the bactericidal properties of the tissue prevail against the destructive action of the microbes, the ulcer begins to clear up, the shreds of necrosed tissue are thrown off, and the epithelium of the sides extends gradually over the floor or bottom of the affected areas, which becomes more or less bright or polished in appearance. The irregular projections disappear during the reparative process. The infiltrated area becomes of a uniform gray tint. Vessels may develop at the borders and at the bottom of the larger ulcers. The diffuse œdema of the cornea subsides. The symptoms of reactive inflammation and irritation sensibly diminish, while the loss of substance is being made good. In brief, the ulcer clears, the grayish floor shines like a mirror, and the lacuna or gap caused by the loss of the necrosed material is replaced by new tissue. The ciliary pain and injection, photophobia, and lacrymation diminish. The pupillary contraction disappears, or at least the iris reacts more readily to atropine.

The vascularity of the sides and of the bottoms of ulcers favors the

nutrition of the tissues, and aids in the limitation of the morbid process. Some of these new vessels proceed from the superficial corneal branches and others from the deep vessels. The latter predominate, however, when the ulcers are of the deep variety.

3. *Cicatrization of the Ulcer.*—The ulcer becomes uniformly covered with tissues that are designed to replace those that are already lost. The tissue used in the process of repair is at first cellular, and is later transformed into fibrillary cicatricial tissue, which is covered with a layer of nearly normal epithelium. Nevertheless, the new connective tissue is always distinguishable under the microscope from normal tissue by the irregular arrangement of its fibrils. This is the more pronounced where the loss of substance has been considerable, especially when the ulcer has been deep. The resulting maculæ of the cornea are more or less apparent according to the extent of the ulcer and the length of time it has remained vascular. (See *Corneal Maculæ.*) The vessels gradually disappear, though some of them may persist in dense cicatrices. Small ulcers, especially in the cornea of children, may disappear without leaving any maculæ. Ulcers of long duration sometimes do not fill entirely, and thus leave behind a corneal depression. This thinned portion is always liable to bulge slightly by the pressure exerted upon it, but rarely sufficiently to reach the surface of the cornea. The ectatic cicatrix is thus liable to develop into a keratectasia.

4. *Keratocele.*—The membrane of Descemet is distinguished from the rest of the corneal tissue by its greater power of resistance to injurious agencies. When the ulceration has destroyed the corneal tissue just in front of this membrane, it yields to the intra ocular pressure and bulges forward, remaining transparent and becoming only slightly or not at all affected. Its free movement upon the overlying parts may cause it to project above or in front of a very narrow ulcer, which then becomes more or less filled by a transparent and mirror-like vesicle. Keratocele usually terminates in perforation. The shreds of membrane that are intercalated in the ulcer retard the formation of a firm cicatrix.

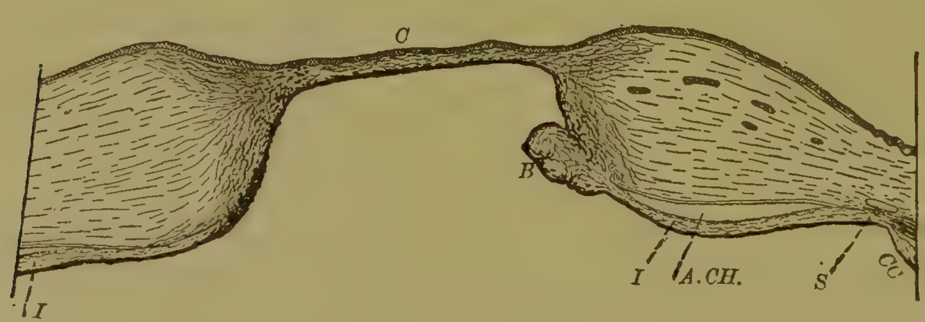
5. *Perforation.*—An ulcer may perforate with or without a preceding hernia of the membrane of Descemet. This often takes place in consequence of some effort which increases the intra-ocular pressure, such as coughing, sneezing, or stooping. The patient usually feels a sudden sharp pain, which is followed by a gush of hot liquid. The aqueous humor runs out, the iris and the crystalline lens are brought close to the posterior wall of the cornea, thus abolishing the anterior chamber, and the tension of the eye is very much lowered. After perforation the symptoms of keratitis generally abate, the pains gradually diminish, and the ulcer approaches the stage of regression. The diminution of the intra-ocular pressure favors the interstitial circulation of the nutrient secretions of the cornea. The opening is closed by the pressure of the iris or the crystalline lens, according to its situation, and it may be further obstructed by the extension of a plug

of fibrin from the iris. This fibrin becomes infiltrated with young cells, and the occlusion is more greatly strengthened by epithelium. Meanwhile the aqueous humor reaccumulates, gradually filling the space between the cornea in front and the iris and the crystalline lens behind. These organs are thus pressed back to their normal positions. Such favorable results following perforation are not the rule, except in the case of small ulcers of the cornea. Persistent adhesion of the iris to the corneal cicatrix is termed anterior synechia.

6. *Hernia of the Iris.*—The iris may become more markedly engaged in the opening, being pushed forward by the aqueous humor. Indeed, this occurs in the majority of rather large ulcers. A hernia or prolapse of the iris is thus produced. The pressure of the aqueous humor tightly holds the iris in place, which thickens and forms a grayish-brown prominence on the surface of the cornea. A layer of fibrinous exudate ordinarily covers the prolapsed iris. As the prolapse of the iris increases, it is held in place more and more securely, and is drawn, with the pupil, towards the perforation. Rupture may take place by the vesicle giving way; but ere long the distention recommences. The tissue of the iris is transformed into a red knob that is almost covered with granulations. This reinforcement by young, fresh tissue may lead to a sinking in of the prolapse, or to a corneal cicatrix with anterior synechia. If the perforation be in or situated near the periphery of the cornea, the synechia is drawn towards it, displacing the pupil. A central perforation, especially when somewhat large, produces a total synechia, involving the entire pupillary margin. The pupil thus may be caused to disappear. If the opening be extensive, the crystalline lens may be expelled at the time of the perforation.

7. *Staphyloma.*—An extensive prolapse of the iris may become cicatrized as a thin fibrillary membrane which is pigmented posteriorly and covered with epithelium anteriorly. An ectasis is produced by the aqueous humor pushing the cicatrix forward. (Fig. 12.) More of the cornea gives way under the pressure, and a staphyloma, which consists of the prolapsed iris, cicatrized on its surface and covered over with epithelium, is produced. The staphyloma may also result from a very narrow perfora-

FIG. 12.




Large cicatrix of the cornea, with thin walls and with adhesions to the iris. To the right and left are stumps of corneal tissue that are thickened by œdema: *C*, corneal cicatrix, formed principally by the iris, and pigmented on its under surface; *S*, canal of Schlemm; *C.C.* ciliary body; *I*, iris; *A. CH.* anterior chamber; *B*, semicylindrical prominence formed by the iris.

tion in which its borders are considerably thinned. They are unable to resist the intra-ocular pressure and are pushed forward with the iris, which firmly adheres to them. In this case the covering of the staphyloma is mainly composed of corneal tissue. This contingency is greatly to be feared when there is a large anterior synechia, and especially when there is a total synechia of the pupillary border. The aqueous humor being no longer able to flow into the irido-corneal angle, the eye becomes hard and glaucomatous. This contributes to the bulging of the cicatrix. A staphylomatous eye is either glaucomatous from the first or soon becomes so.

8. *Glaucoma.*—A cicatrix with an anterior synechia may be so firmly fixed as not to become ectatic. If, however, the synechia is large, and particularly if there be total synechia of the pupil, the eye will be ultimately lost from glaucoma. Finally, the traction on the iris and the deep infection of the eye may lead to an iridocyclitis, a phthisis, or a panophthalmitis, or to intra-ocular hemorrhage that may disorganize the eyeball.

9. *Fistula of the Cornea.*—An opening made at the time of perforation may remain patulous for days or even weeks. In such a case a true corneal fistula has not been produced, but a fistulous staphyloma, which will be considered later in connection with a description of the other varieties of staphylomata.

*Etiology.*—The chief predisposing cause of corneal ulcers is the absence of blood-vessels in the cornea, which are so efficient in the struggle against the microbes. The contrary holds good in small erosions and infiltrations of the conjunctiva, that generally heal quickly because of rich vascular supply. The more direct causes of ulcers are of two classes: 1, traumatic; 2, microbic. (See *Superficial Traumatic Keratitis.*) Usually both of these causes are required to produce an ulcer, especially one which lasts any period of time. As has already been stated, the freedom of infection by pathogenic organisms enables the ulcer, which then really does not deserve this name, to heal rapidly.

1. *Traumatic Causes.*—(*a*) It is not necessary to mention here all the ways in which injuries may give rise to a superficial keratitis. (See *Superficial Traumatic Keratitis.*) Traumatisms that persist or are often repeated generally result in the formation of ulcers. An important cause favoring the repetition is the non-occlusion of the eye, either in consequence of insufficiency of the lids, congenital or cicatricial, or because of the insensibility of the eye from partial or general trigeminal paralysis. Winking need not be abolished, as it may still occur through the influence of the sensibility of the other eye. Particles of dust striking an eye without sensibility no longer provoke the reflex winking and lacrymation that are necessary to eject them. If winking is diminished or suppressed, the inflammation may assume the character of a neuroparalytic keratitis. Grave affections that lower the general sensibility, such as enteric fever or typhoid states, have usually the same effect. The same is true of variola, in which condition the corneal erosions are in addition infected.

(*b*) Among traumatic causes conjunctival and palpebral lesions, such as granulations and cicatrices of the conjunctiva and of the ciliary border, absence or deviation of the cilia, and irregularities of the conjunctiva of the upper lids, are all of especial importance. Conjunctival catarrh rarely produces ulcers of the cornea, except in aged people. They occur most frequently under the form of peripheral growths that commence as infiltrations. Blennorrhœa acts in two ways,—by the friction of the turgid, puffed conjunctiva against the cornea, producing superficial exfoliation, and by compressing the nutrient vessels of the cornea. The pus is, moreover, a cause of infection. This is likewise the case with diphtheritic conjunctivitis, in which the diphtheritic and other microbes infect the traumatic superficial exfoliations.

(*c*) All the lesions that produce superficial keratitis, such as corneal phlyctenules, conjunctival herpes, vesicular keratitis, etc., may become the starting-points of very serious ulcers. In many of these cases of superficial keratitis small ulcers form, which, though infected, have not the tendency to progress. To bring this about, a special cause of infection seems to be necessary. Maladies usually described as superficial may have already become of serious import by the development of ulcers, such as are seen in dendritic keratitis, rodent ulcer, and herpes zoster of the cornea.

2. *Causes of Infection.*—The cornea may become infected from blennorrhœa, diphtheria, or secreting granulations of the conjunctiva. Ulcerous and non-ulcerous blepharitis and blennorrhœa of the lacrymal sac are frequent sources of infection, as well as eruptions of the face, eczema, pimples, coryza, and, above all, ozæna. Infectious germs may by the hands or a handkerchief be conveyed to the eye from eruptions, furuncles, wounds, etc., situated on any part of the body, and especially from those affecting the nose. Many of the agencies causing the traumatism, such as particles of earth, stone, blades of straw, hay, etc., infect the cornea while wounding it. (See also *Suppurative Keratitis.*)

*Prognosis.*—As to the effect on vision, the prognosis in cases of ulcer is very variable, and can be made only by a judicious estimate of the numerous factors which have been previously specified. The influences that contribute to produce this result are the locality, the depth and size, and the degree of infection of the ulcer. A peripheral ulcer of small size produces a macula which only slightly interferes with vision. On the other hand, sight is diminished or even abolished if the macula encroaches on the pupil. (See articles on *Corneal Macula* and *Corneal Staphylomata.*)

*Treatment.*—There are few diseases in which appropriate and judicious treatment exercises so favorable an influence as in ulcers of the cornea. Exciting causes of injury should be diligently searched for and eliminated as far as possible. Cilia which rub against the cornea and also papillomatous excrescences of the conjunctiva should be removed. Sojourn in a dusty atmosphere or in the rain, etc., should be interdicted. Foci of infection, such as blepharitis or blennorrhœa of the lacrymal sac are to be de-

stroyed, or at least abated,—the blepharitis by antiseptic measures, and the diseases of the lacrymal passages by sounds and boric acid injections. Any eruption of the skin on the face or elsewhere and all nasal affections should receive appropriate treatment. Catarrh of the conjunctiva, whether it has preceded or has been caused by the ulcer, should be treated, with careful avoidance of remedies that might irritate the diseased cornea. Boric acid lotions should be used frequently. The palpebral conjunctiva may be cautiously touched with nitrate of silver, the cornea being well protected and the effect of the caustic neutralized with chloride of sodium. The indications for treatment in ordinary cases may be summed up to be, the use of antiseptic lotions, instillations of atropine, and the use of a bandage. The occlusive and compressive bandages play leading *rôles* in the treatment of corneal ulcers. They immobilize the lids and prevent them from irritating the ulcers, and thus often allay superficial pain. They protect the ulcer from dust-particles, which winking would not be able to remove from the excavations. They prevent the free border of the lids, this receptacle for microbes, from infecting the ulcer. When compressed a little more firmly they support the base of a deep ulcer, preventing it from yielding and perforating. (See *General Pathology.*)

A profuse secretion, as in cases of blennorrhœa, diphtheria, or granulations, may be a contraindication to the employment of the bandage. The reason for this is that the retained secretions undergo decomposition, and thus infect the ulcer.

*Atropine.*—The employment of atropine can seldom be dispensed with. The indication for its use is given by the signs of congestion of the iris,—ciliary pain, contraction, and sluggishness of the iris tissue. Dilatation of the pupil immediately alleviates ciliary pain and causes improvement in the appearance of the ulcer. Furthermore, the drug prevents the formation of posterior synechia and breaks recent adhesions. After the healing of the ulcer the synechia may impair vision and produce glaucoma. The contraindications resulting from the presence of large anterior or posterior synechiæ must not be forgotten. Every time that atropine fails to dilate the pupil it tends to render the eye glaucomatous, and in such a case should not be employed. In such a case the writer would prefer instillations of *eserine* or, rather, of *pilocarpine,* which is less painful, three or four times a day. Some authors recommend miotics in all cases of corneal ulcer. This is done for the purpose of diminishing the pressure in the anterior chamber, especially when there is danger of perforation. Instillations of miotics are disliked because they all (especially eserine) produce ciliary pain and favor the formation of synechia by maintaining the iris closely against the crystalline lens.

*Antiseptics.*—Corrosive sublimate solution or a solution of cyanide of mercury should be liberally used one or more times daily in cleansing the conjunctival sac and the ciliary margins of the lids. Lotions of this kind, combined with atropine and followed by a bandage, suffice in most cases.

A greater degree of infection, indicated by marked infiltration, violent reaction, extreme congestion of the iris, and rapid progress of the ulcer, however, demands the employment of more energetic and more lasting antiseptics. Iodoform insufflated under the lids, followed by a bandage over the eye, has an action that is less intense, but more lasting. The conjunctival secretion is also slightly diminished by its use. Applications of the crayon of pyoktanine (preferably the methyl blue) over the bottom and the infiltrated margin may produce very marked amelioration in the appearance of the ulcer.—In cases of intense infection and rapid progress, cauterization by heat (see vol. iii. p. 823) is the sovereign remedy, and is usually very efficacious. The floor and especially the infiltrated borders of a reddening ulcer, which is progressing rapidly, must be cauterized. Perforation of the cornea should be avoided, although the mischief caused thereby will be minimized through a chilling of the cautery by the aqueous humor. A distinct cicatricial macula will remain upon the healing of such an opening.—Instillations of cocaine should precede all applications of the cautery or of the crayon of pyoktanine to the cornea; it may even be advisable to instil it before making a simple examination of the eye or applying a lotion. It has no beneficial influence on the ulcer itself.

*Paracentesis.*—If spontaneous perforation (the happy effects of which have already been described) seems likely to occur, it is better that the opening should be made by the surgeon, preferably in a healthy portion of the periphery of the cornea. Hernia of the iris and other grave accidents are more easily avoided when paracentesis is done in normal corneal tissue. Absolute rest of the patient should be enjoined whenever perforation is threatened.

If there be perforation without notable attachment of the iris, ordinary treatment is sufficient. A true prolapse of the iris should be released after cocainization either by excision or by the actual cautery. When the prolapsed iris slips away in front of the scissors, it is necessary to pierce and incise it laterally by means of a von Graefe or a Beer knife, and then to excise it with a scissors. Excision and cauterization are, however, of little use if the prolapse be very large, but it may be made smaller by repeated punctures.—An iridectomy which re-establishes access of the aqueous humor in the anterior chamber is one of the best means of flattening a prolapse and of obtaining a firm, resisting cicatrix.—In a central prolapse of moderate size, atropine acts favorably by drawing the iris towards the periphery; if the prolapse be a large one, this drug is not only useless, but tends to augment it. The latter cases call for the use of eserine, which acts very favorably in peripheral prolapses that are not exceedingly large. The action of this drug is to flatten them sensibly by augmenting the resistance of the iris.—In those synechiæ in which there is a wedging in or an enclosing of the iris, it has been recommended (A. Meyer, etc.) to cover the perforation (after the edges have been curetted) with a fragment of conjunctiva that has previously been detached and drawn over the opening by means of a suture.

This operation is especially useful if the iris reaches the surface, thereby exposing it later on to infection. The conjunctiva adheres to the ulcer and reinforces the cicatrix.—A keratocele should be pierced or tapped. (Concerning the sequelæ of ulcers, see *Maculæ* and *Staphylomata of the Cornea.*)

Irritant remedies may be cautiously tried, if the period of progression tends to be prolonged and if there are corneal facets. The mildest of these consist in the application of hot compresses. Calomel is more energetic; and still more so is the yellow oxide of mercury ointment, employed as in phlyctenular disease. When an ulcer has lasted for a long while and has become indolent, scraping of its bottom and sides with a small metallic spatula is often attended with most beneficial results.

(*b*) **Suppurative Keratitis; Suppurative Ulcer and Abscess of the Cornea.**—The gravity of the situation is augmented in keratitis by copious suppuration, which tends largely to destroy the cornea, and to produce perforation, staphyloma, etc.

1. *Ulcer and Abscess.*—Suppuration may follow a traumatic loss of corneal substance and render a simple ulcer purulent. A keratitis may be openly or freely suppurative at first, but more frequently suppuration commences later by a dense, cellular infiltration of the tissue of the cornea, which it softens and destroys. Before the central softening is completed the infiltration ulcerates through the surface, exposing the infiltrated and partly necrosed corneal tissue. It never forms an abscess in the usual signification of the word,—a cavity filled with pus, discharging its contents later through a narrow opening,—even when the infiltration has been primarily deep. Some authors use the word abscess when the sides or borders of an ulceration are undermined or are hollowed out, constituting sinuses for the pus. The border is then more opaque than the centre. Other authorities apply the term abscess to ulcers which have a very irregular base or floor on which shreds of necrossed tissue remain for some time before being eliminated.

2. *Symptoms.*—A purulent infiltration possessing no opening for the escape of the accumulated pus may vary in its degree of intensity and size. It is important to be able to recognize an abscess which has a tendency to produce wide and deep destruction of tissue. If it be small and superficial, there appears a small prominence on the corneal surface; if it be deep-seated, the cornea may only assume an aspect of dulness without any elevation of its surface. In every case it is distinguished by an intense yellowish-white color (not grayish or translucent), which diminishes towards the periphery and merges into a slightly congested œdematous zone, and may be gradually lost in the unaffected portions of the cornea.

A deep purulent infiltration usually increases in size before it ulcerates. The color is an intense yellowish white; where the tint is less intense and grayish, the infiltrations do not tend to suppuration or softening. The œdematous areola ordinarily extends over the entire cornea, which appears dull through its entire extent. The symptoms are violent; there are extreme ciliary and conjunctival injection and iritis, and sooner or later hypopyon

supervenes. Pain may, however, be absent. Quite often there is tactile anæsthesia of the cornea.

The floor of a suppurative ulcer is generally very irregular, and in some places it is infiltrated; the borders may also be infiltrated and constitute pockets in which the pus stagnates. It is useless to subdivide suppurative ulcers any further, though the *ulcus serpens* (Saemisch) or serpiginous ulcer merits special mention. It is rather large, is situated in the central portion of the cornea, and develops only through infiltration of one side. In addition, the epithelium may even advance over or towards the ulcer, which becomes elliptical. There is hypopyon, with a tendency to colliquation of the cornea.

According to Uhthoff and Axenfeld, the pneumococcus is the exciting agent in nearly all cases of serpiginous ulcers of Saemisch. This organism is normally present in the nose, the throat, and the lacrymal ducts, and takes an active part in inflammatory lesions of these regions. It is, therefore, easy to understand how it gains access to the eye with the greatest ease.

3. *Hypopyon.*—Certain authors speak of a suppurative keratitis only when there is hypopyon,—that is, when there is pus in the anterior chamber. The pus on mingling with the aqueous humor sinks to the bottom of the anterior chamber, where it forms a yellowish-white collection, which is horizontal or even slightly concave on top. Most frequently the composition of the pus is compact and fibrinous; rarely it is liquid, and then it is displaced when the head is inclined in different positions. Where the hypopyon is small, it is called an "onyx," because of its resemblance to the root of the nail. The amount may be so insignificant that it is concealed behind the limbus conjunctivæ, and can be discovered only by examining the eye obliquely from above downward. The pus may reach the level of the pupil or even fill the anterior chamber. Hypopyon is an exceedingly grave complication in a case of keratitis. Although a small hypopyon may disappear by resorption, a large one, invading the major part of the anterior chamber, tends to produce necrosis of the cornea and destruction of sight in the affected eye.

The pus of hypopyon issues from the irido-corneal angle, from the iris, and; in severe cases, also from the ciliary body, from whence it flows across the pupil. It was for a long time supposed that hypopyon was a corneal abscess which had opened posteriorly, or that the pus-cells passed through the interstices of the posterior corneal lamellæ (Horner). It is true that yellowish pus-tracks on the posterior face of the cornea may be seen extending downward from the level of the ulcer to the hypopyon. These are composed of filaments of fibrin, impregnated with cells, which are deposited by preference behind the ulcer, in which situation the endothelium is altered and may even be elevated or lifted off (Leber). Experimental infection of the cornea with pyogenic organisms made upon animals shows that in hypopyon the posterior planes of the cornea may be intact and even

remain free from cellular infiltration. Fig. 13 is a cross-section of an eye affected by a serpiginous ulcer (of Saemisch) of moderate severity, which would probably have healed if the patient had not died of an intercurrent pneumonia. The deep corneal planes are intact and without cellular infiltration. The fibrinous hypopyon extends to the inferior border of the dilated pupil. It is infiltrated by cells, especially in its anterior planes. Its posterior portion is composed of filaments of fibrin, that were probably furnished by the iris. The cellular deposits seem to issue from the irido-corneal angle, or perhaps from the pupillary margin of the iris. A thin layer of pus exists in the posterior chamber, which is most conspicuous in its lower portions; it extends throughout the ciliary body as far as the ora serrata, where it penetrates the vitreous humor, as shown by the small abscess at ($A$) in the illustration, and by a little accumulation of pus ($P$) situated behind the crystalline lens. The iris and the ciliary processes are infiltrated by a homogeneous exudate and their vessels are markedly congested. The periphery of the iris is more or less adherent to the cornea.

FIG. 13.


Corneal ulcer with hypopyon (ulcus serpens); occurring in a case of fatal pneumonia (Nuel). *I* and *E*, limits of the corneal ulcer; *I*, area of infiltration at the edge of the ulcer rendering it progressive; *H*, hypopyon; its lower portion consists of a deposit of pus, which also permeates the whole of the ciliary body; *P*, deposit of pus-cells behind the crystalline lens; *A*, small abscess of the vitreous body, the pus of which has extended from the ora serrata; *Z*, ora serrata; *F*, free parenchyma and lamellæ of the vitreous body; *R*, anterior limit of the retina proper.

Hypopyon, therefore, may transform a simple inflammation of the cornea into a keratitis complicated with grave iritis, or even into a cyclitis,—that is, into a disease of the whole anterior segment of the eye. This explains the long duration of the malady and the persistent irritation, even when the ulcer presents an improved appearance. Still it is to be supposed that in cases of slight hypopyon the suppuration does not extend behind the iris to the same degree as it does in severe ones.

Similar cases have since been described anatomically by Uhthoff, Axenfeld, and others. According to Elschnig, a perforation of the membrane of Descemet has been found by Verdese, Green, and Ewing in cases where the corneal tissue proper was not yet perforated. This point needs further investigation. It is supposed that the migratory cells which have accumu-

lated in front of Descemet's membrane corrode its tissue by histolysis, just as they do in detaching the necrosed parts of the fundus of an ulcer.

There are no microbes in an hypopyon like that shown in Fig. 13, the deposit of pus within the eye being induced by the irritating action of the bacteria found on the cornea. We admit that phlogogenic substances secreted by the intracorneal microbes (in cases of non-perforation) diffuse themselves in the pericorneal vessels, where they provoke dilatation of the pericorneal vessels and emigration of migratory cells. In the same manner the phlogogenic substances are diffused in the anterior chamber and the iris, and, if they reach this membrane in sufficient quantity, they give rise to iritis and hypopyon, the latter being an exudation from the iridian vessels. When bacteria are present in an hypopyon, the affection has a tendency to develop into panophthalmitis. This is likely to happen after perforation of the cornea.

The purulent secretion on the surface of the diseased eye comes partly from the cornea and partly from the conjunctiva after the latter has become infected.

Suppurative keratitis on healing will leave a dense macula even when it is arrested before perforation occurs. After perforation has taken place, all the consequences resulting from ulcers may be observed. Staphyloma is most to be feared.

Anterior polar cataracts,—distinct from pyramidal cataracts,—consisting of small, well-defined, circular, nacreous, white spots, not projecting into the anterior chamber, and which are usually considered to be of congenital formation, are generally formed in extra-uterine life, and usually follow ophthalmia neonatorum complicated with ulcus corneæ, with or without perforation of the membrane.—The phlogogenic substances above mentioned, secreted by the intracorneal microbes, diffuse themselves (in cases of non-perforation) in the aqueous humor and set up iritis. At the pupil they come in contact with the lens capsule, which is here not protected by the iris, and penetrate to the capsular epithelium. The epithelial cells, which are still in full formative activity at this age, are irritated and proliferate, forming the laminated tissue that constitutes the cataract. At a more advanced period of life non-perforating corneal ulcers (unaccompanied by hypopyon) never give rise to the same disturbance of the lens, partly because the capsular cells are relatively inactive and partly because the anterior chamber is deeper. (Nuel.)

4. *Etiology.*—Suppurative keratitis is always the result of severe microbic infection, which is either due to the presence of a larger number of microbes than in a non-purulent ulcer or because the bacteria themselves have assumed a greater virulence. At times the resistance of the corneal tissues seems to be diminished, owing to the existence of a grave general malady or a contusion of the cornea. The infection always comes from without, through a loss of substance, which may be sometimes insignificant, of the surface of the cornea. The writer regards as erroneous the belief that endogenous (metastatic) infection may occur in suppurative keratitis supervening in infectious diseases, such as variola, typhus, scarlatina, and all maladies of a typhoid character, like pneumonia.

There must always be a cause of injury and a source of infection. As

to causes of injury, it is only necessary to recall what has already been said of ulcers in general. In wounds or contusion of the cornea, the microbes in the conjunctival sac appear to be sufficient to provoke suppuration. The body wounding the cornea may be infected at the time of injury. Bits of earth or stone, the beard of wheat, and fragments of straw seem to be capable of producing infection, as shown in the keratitis of reapers. Three-fourths of ulcers with hypopyon have their source of infection in diseases of the lacrymal passages, which should, therefore, always be examined in cases of this kind. Even in cases of simple obstruction of the lacrymal ducts, the secretion, which is held in the lacrymal sac until it flows back into the conjunctival sac, contains the bacteria of suppuration, such as the staphylococcus, the pneumococcus, and, at times, the streptococcus. It is the same with the nasal secretions, in cases of rhinological ailments, especially in ozæna. The conveying of the secretion (through the fingers, the handkerchief, etc.) to the eye is quite easy of accomplishment. It would seem also that, in the case of diseases of the throat, the secretion of this part of the body may infect the eye in like manner. A frequent cause of serious corneal infection is to be found in furuncles and in all eruptions that are situated near the eye. Conjunctival maladies, diphtheria, blennorrhœa, and secreting granulations may also be cited as causes of suppurating corneal ulcers.

In about two per cent. of the cases of variola, abscess of the cornea supervenes most frequently during the period of desquamation. The eruption of pustules upon the cornea in such a case must not be considered as having the same origin as that upon the skin. A patient affected with small-pox, for example, is unable to close the lids, and the open eye is bathed with pus and fragments of crusts.

Exceptionally, a variolous papule may develop on the conjunctival limbus. Conjunctival diphtheria, which so often covers the whole corneal surface with pus, extends only exceptionally to the cornea. Formerly the involvement of the cornea was considered to be caused by necrosis due to lack of nourishment. At present the opinion is expressed that it results from pyogenic microbes penetrating through the small corneal sores, which must of necessity occur in conjunctival diphtheria. According to H. Coppez, the entrance of bacterial toxines into the corneal tissue should also be taken into consideration as a factor in the production of this condition. Even though gonococci have been found in the corneal lamellæ of suppurative keratitis following gonorrhœal conjunctivitis, it would seem that this, also, is a case of infection of the cornea (through small ulcers) by pyogenic microbes. The progress of the diphtherial and gonorrhœal infection appears to be favored by compression of the superficial ciliary vessels.

In isolated cases ulcerous and purulent keratitis has been caused by mycelia from fungi that have invaded the tissue proper of the cornea (Leber, Schirmer, and others).

5. *Prognosis.*—The prognosis in suppurative keratitis is always serious. First of all, any part of the cornea that is invaded by the purulent process is sure to be, under the most favorable circumstances, opaque after cicatrization. Frequently, after the disease is cured, posterior synechia will

remain; and if they be numerous, they destroy the sight and predispose the eye to glaucoma. The latter condition may necessitate an iridectomy. It is not rare to find, later on, that the crystalline lens has grown opaque (cataract). The greatest danger that threatens such eyes is the presence of corneal staphyloma, the tendency of which is to destroy the sight. (See *Corneal Staphyloma*.) In the case of perforation a staphyloma is greatly to be feared, on account of the softening and the destruction of the corneal tissue. The greatest number of staphylomas result from suppurative keratitis. Sometimes the infection invades the depths of the eye and leads to panophthalmitis.

6. *Treatment.*—The treatment is the same as that of grave corneal ulcers. In mild cases ordinary measures may suffice (see *Corneal Ulcers*), but extensive suppuration calls for the employment of the most energetic bactericidal remedies with cauterization by heat (see vol. iii. p. 823). Experience has proved that when an hypopyon has reached the inferior border of the pupil, resorption of the pus is no longer possible. It must then be evacuated by paracentesis. This can sometimes best be accomplished by the use of forceps or by injection of the physiological solution of salt. If possible, the puncture should be made below the level of the pus into a portion of the cornea which is still healthy. In large ulcers which are threatening to perforate and in grave ulcus serpens the original incision recommended by Saemisch (see vol. iii. p. 827) may be made. This method has been less often resorted to since the introduction of antiseptic measures, and, above all, of cauterization by heat. The anterior chamber is opened with a von Graefe knife through a portion of the unaffected cornea near the ulcer, and a counterpuncture is made in the healthy cornea on the opposite side of the ulcer. The two openings are then connected by an incision extending through the greater diameter of the cornea. The pressure on the posterior side of the cornea is diminished, and the outflowing aqueous humor carries away the pus. The borders have a tendency to become agglutinated, and should be daily reopened. If the case progresses favorably, the result will be a cicatrix with a large anterior synechia.

In a large hypopyon the employment of atropine is without beneficial effects. Its use augments the danger of perforation, and the paralysis of the iris and the synechiæ prevent the pupil from dilating.

Lavage of the anterior chamber with antiseptics has not given good results. Corrosive sublimate solutions, particularly, are exceedingly irritating, and all of the other salts of mercury *per se* give rise to a persistent opacity of the entire cornea.

During the last few years, subconjunctival injections of the bichloride of mercury have been tried (Darier, Abadie, de Wecker, and others). After cocainization, several drops or even larger amounts (de Wecker) of the corrosive sublimate solution of the strength of 1 to 2000 or 1 to 1000 are injected beneath the conjunctiva. For the same purpose Chibret recommends the cyanide of mercury, as less irritating than the corrosive subli-

mate. He uses half a syringeful of an aqueous solution of the cyanide of mercury in the strength of 1 to 200, or even 1 to 100. Though this treatment appears to be founded on rational principles, especially as Pflüger has shown that substances thus injected beneath the conjunctiva really reach the cornea, it does not seem to have given better results than when the antiseptics are applied by other methods. Indeed, Bachleur, from his experiments on rabbits, disputes their efficacy. In addition, the injections are attended with considerable pain, as the cocaine is not capable of rendering an inflamed eye insensible. One can thus understand how the first enthusiasm for this mode of treatment has disappeared, and how the tendency at the present time is to abandon its employment.

Injection (beneath the conjunctiva) or instillation of anti-staphylococcus or anti-streptococcus serum has not yet been of real benefit.

*A priori*, the use of wet corrosive sublimate compresses would appear to be of use in bringing the cornea in continuous contact with this antiseptic. Though often employed, they do not seem to produce markedly favorable results. This is due, in the writer's opinion, to the secretion from the conjunctival sac not being absorbed by the material employed for the compress when it is in a moist state.

The rule is to use dry compresses, which are to be renewed two or three times each day, at which time the eye is to be thoroughly cleansed with the solution of corrosive sublimate or of cyanide of mercury. When iodoform has been sprinkled on the compress, lavage with the bichloride of mercury solution must not be practised; the solution of the cyanide of mercury or of boracic acid can, however, be employed along with the iodoform.

Atropine is often used, care being taken to watch carefully its action upon the pupil: if it dilates, its effect is relatively beneficial. If it does not do so, as in the case of a large hypopyon, the action of the mydriatic is injurious. (See *Treatment of Corneal Ulcers.*) In these cases the use of miotics is often painful and unattended with good results.

Warm compresses, applied for an hour at a time once or twice a day, often do good, as they favor the interstitial nutrition of the cornea.

In every case care must be taken to insure cleanliness of the eye by daily repeated lotions of the conjunctival pocket with mild antiseptic solutions of sublimate, boric acid, etc.—In cases of great conjunctival secretion the application of iodoform may be useful.

(*c*) **Keratitis with or from Lagophthalmos; Neuroparalytic Keratitis.**—The forms of keratitis designated by these names have the characteristic in common of being primarily dependent upon injuries of the cornea due to the defective action of the lids. Secondary invasion by pathogenic organisms naturally cause these lesions to assume a more serious type. They are in the main treated of in the preceding pages; some in connection with corneal ulcers, the others with suppurative keratitis. Neuroparalytic keratitis is usually described separately, partly because of its clinical features and partly because of its supposed etiology and pathogenesis. Keratitis

with lagophthalmos is considered here because it is mainly due to the same cause as neuroparalytic keratitis, and in time becomes converted into this type of keratitis.

*Keratitis with Lagophthalmos.*—The causes are the same as those enumerated in the preceding pages; everything which obstructs or prevents the normal occlusion of the lids, ectropion, insufficiency or shortening of the lids, either congenital or acquired, from cicatrices, exophthalmos, retrobulbar tumors, Graves's disease, etc.

Paralysis of the facial nerve acts in the same way. When thus exposed, the normal cornea, especially its lower segment, desiccates and exfoliates, and the conjunctiva becomes irritated. During sleep, the reflex-winking being suppressed, the eye remains more or less open. During the day the affected eye hides itself under the upper lid, which does not wholly perform its normal function of removing the foreign particles that collect upon the cornea. Traumatic ulcers that may become infected, and which are situated most frequently in the inferior half of the cornea, may result.

Keratitis associated with a typhoid state has an analogous origin, though it more nearly approaches the neuroparalytic form.

*Treatment.*—The main points to be attended to are: (*a*) the protection of the eye with a bandage, which may have to be worn even at night; and (*b*) the use of antiseptics. Cicatrices in the region of the eye sometimes call for operative measures.

*Neuroparalytic Keratitis.*—Trigeminal disturbance, which is always more or less anæsthetic in type, may be accompanied by herpes zoster of the cornea. Though this condition has probably a special pathogenesis,—a neuritis extending to the cornea,—it will be considered in this connection, owing to its close relation with the matter now under discussion.

True neuroparalytic keratitis is observed in complete paralysis of the trigeminus or in one of its ocular branches, and especially the nasal branch. Under these conditions the reflex action of winking and of lacrymation does not take place. The eye ordinarily remains open, its surface becoming dry in the same manner as if it had been cocainized. The eyeball—especially the cornea—is no longer protected by that guardian of the eye, the trigeminus, and is exposed to contusions, foreign bodies, and desiccation by evaporation of what little moisture there may be there, the secretion of tears having been already suppressed. Thus the conjunctiva becomes irritated, and the cornea exfoliates more and more. Sooner or later the wound becomes infected and the infiltration extends in depth. Usually the centre of the cornea is especially involved, the extreme periphery of the cornea remaining relatively undisturbed. Suppuration now takes place and an hypopyon is produced. The picture of a typical case is completed with perforation, loss of tissue, staphyloma, and phthisis bulbi. In favorable cases cure is effected with a large central leucoma, which later on may permit of the performance of an iridectomy.

The theory of neuroparalytic keratitis dates from Magendie, who saw suppurative keratitis supervene in rabbits in which he had divided the trigeminal nerve within the cranium. It is supposed that there are in the trigeminus trophic nerve-fibres which preside directly over the nutrition of the elements of the cornea, very much as the motor nerves act on the muscles and the secretory nerves on the gland-cells. In pathology trophic nerves are still spoken of in the sense indicated, but their existence has not yet been clearly demonstrated. As regards the experimental evidence, Snellen points out that the insensibility of the eye means a great deal in regard to the production of this variety of keratitis; an animal hits and injures its eye because it no longer feels the hurt. Feuer also calls attention to the desiccation of the cornea which results from the absence of winking after the eye has become insensible. Eberth, furthermore, invokes the liability to microbic infection of an eye thus exposed.

There is no necessity of having recourse to the trophic hypothesis in explaining the etiology of neuroparalytic keratitis. The explanation of Snellen, that the insensibility of the eye enables the usual causes to act without obstacle, appears to be the true one, though he did not at the time of advancing this theory know of the important part taken by bacteria in the production of this condition. It is certain that the suppuration, often advanced as being characteristic and demonstrative of the neuroparalytic nature of this disease, cannot result alone from paralysis of trophic nerve-fibres. The seat of the disease being in the centre of the cornea and not within the inferior half, as in some cases of lagophthalmic keratitis, does not suffice for the making of an essentially separate morbid form (neuroparalytic) of it. It is true that the entire eye is insensible and that the palpebral opening is largely increased, thus exposing the cornea to injurious influences. It is more natural that the centre of the cornea should be affected, as this portion of the eye is the most remote from the blood-supply. In non-anæsthetic keratitis with lagophthalmos, the upper lid generally covers the cornea more or less. The patient is thus enabled to turn the eyeball upward and remove by this action any foreign particles which may be present upon the cornea. Reflex lacrymation is not suppressed. Even now many authors attempt to demonstrate the neuroparalytic nature of this disease by basing their assertion upon the fact that suppuration may sometimes begin in the depth of the eye, in the iris, while at that time the cornea may still be intact. This very point, however, argues against their interpretation of it. Moreover, it is more than probable that the integrity of the cornea is more apparent than real. The neuroparalytic hypothesis is accepted by most of the present writers. They base their opinion upon a single experiment of Merkel, in which a keratitis in a rabbit was produced by the ascending fibres of the trigeminus alone being injured. The cornea remained sensible, and it is, therefore, asserted that the ascending fibres of the trigeminus are trophic in character.—The writer admits that vasomotor disturbances resulting from paralysis of the trigeminus might favor the outbreak of keratitis (Claude Bernard), but does not believe that they alone are capable of producing it. An alteration of the Gasserian ganglion is particularly apt to be the cause of the paralysis of the trigeminus: the lesion giving rise to this condition may be still more peripheral, as, for example, a neuritis or an operative wound of the filaments of the ophthalmic branch of Willis. At other times the cause for the paralysis seems to be more central than the Gasserian ganglion and to have its location in the medulla oblongata itself.—Grave keratitis in typhoidal diseases and keratomalacia in children are more or less comparable to neuroparalytic keratitis. The eyes in typhoid cases are seen to be widely opened, dry, and not protected by winking. This is certainly the result of a nearly complete insensibility of the eye. The same explanation holds good, from one cause or another, in the keratitis that occurs in cachectic children.

*Treatment.*—In considering the treatment for this condition, it is important not to be carried too far by the neuroparalytic theory. Diligent and careful therapy may often save a little vision in an eye which would otherwise be hopelessly lost. Local measures, based on the traumatic theory of the disease, are urgently needed and efficacious. These consist in the use of antiseptics and the application of a bandage. The bandage protects the cornea from most traumatisms, from desiccation, and from infection by pathogenic bacteria. In some cases the bandage may be replaced by simply fastening the eyelids together by means of surgeon's plaster. The dressings are to be renewed many times during the course of the day, and the eye is to be cleansed in a thorough manner by the use of an antiseptic solution. Iodoform is sometimes of value when the conjunctival secretion is marked. The treatment should be continued for a long while,—until the sensibility has partly returned. Even then relapses are to be feared.

If it is possible so to do, the trigeminal paralysis should be treated. For this purpose the iodide of potassium is often indicated. One should not place too much confidence in the application of electricity to the cornea or in the use of hypodermatic injection of strychnine.

*Keratomalacia (simple); Xerophthalmic Keratomalacia.*—Xerosis of the conjunctiva (see article on *Diseases of the Conjunctiva*) may extend to the cornea, which exfoliates and becomes overspread with a similar grayish, silky, and fatty covering. Wide-spread necrosis of the cornea is very apt to occur. There is no true suppuration, no hypopyon, and no pain. The symptoms are due mainly to a xerosis of the conjunctiva, but the corneal affection considerably aggravates the ocular disorder, and indicates that the general condition is seriously compromised. It is a disease of infancy, and results from a cachexia, a species of kerato-conjunctivitis with anæsthetic lagophthalmos, upon which bacteria stamp a special impress. These microbes (Neisser, Leber, Weeks, and others), however, have not been fully identified. In infants, death, after a brief delay, is the rule (Leber); older children may escape a lethal result (Gouvea, Kollock). Upon the evidence of an autopsy made by Virchow in a case of this kind, von Graefe attributed the corneal disease to infantile encephalitis. By others, however, who have made autopsies upon infants so affected (Parrot, Jastrowits), the condition of the brain has been found to be normal.

A pernicious variety of keratomalacia, but one which is not accompanied by xerosis of the conjunctiva, is the so-called "simple keratomalacia." It may be observed in those children whose general nutrition is markedly depressed from whatever cause, such as diarrhœa, typhoid states, etc. Indeed, in some instances it is analogous to the type that is seen in the grave corneal complications of typhoid fever. Here the insensibility of the eye and the denutrition of the cornea afford a more favorable opportunity for microbic infection.

*Treatment.*—This should aim at building up the strength of the patient through alimentation. Locally, antiseptics must be used and the eyes

should be protected by a bandage. The nutrition of the cornea may be stimulated by the employment of hot compresses.

### B.—DEEP, NON-SUPPURATIVE KERATITIS.

The greater number of these affections are due to general constitutional disorders, and if they show the presence of microbes the infection is said to be endogenous. (See *General Pathology.*) As a general rule, the keratitis begins in the tissue proper of the cornea. Such cases are, therefore, analogous to diseases of the fibrous tissues in other parts of the body; and, like them, are of long duration and very often demand general treatment. They show no tendency to produce suppuration or lead the formation of corneal ulcers.

(*a*) **Parenchymatous Keratitis.**—This condition is also known as *interstitial keratitis* and *deep diffuse keratitis.* It is a rather frequent disease, having as its prototype deep non-suppurative keratitis.

*Symptoms.*—A grayish discoloration appears in the periphery or in some other part of the cornea. This change of color soon spreads over the entire corneal membrane, which is infiltrated and its surface dulled throughout. At first the infiltration is translucent, like that seen in œdema of the cornea; it then thickens and becomes of a grayish tint, which deepens until the iris is almost or quite obscured from view. Vision is greatly diminished, and may be reduced to mere perception of light. There is typical ciliary injection, usually without congestion of the conjunctiva. Lacrymation, slight photophobia, and ciliary pain are also present, for the disorder is always accompanied by iritis, or at least by extreme congestion of the iris. The anterior chamber is usually deepened, and the tension of the eye is frequently reduced. In subsequent stages glaucoma may occur.

The disease ordinarily attacks both eyes, either simultaneously or consecutively. When a single eye is attacked the patient should be apprised of the danger to the other. After two or three weeks, sometimes after several months' time, the malady begins to disappear, starting with the periphery after tiny vessels have developed in the depth of the membrane. The clearing process may occupy many months or even a whole year. Examination with a strong magnifying lens under focal illumination will always show the persistence of a certain diffuse opaqueness that is situated chiefly in the centre of the cornea. Vision, though it becomes greatly improved, remains somewhat impaired, as indelible spots of very dense tissue: true corneal scleroses too often remain behind. Such is the classical form of parenchymatous keratitis. Atypical varieties result (*a*) from variations in the thickening process or from a more copious infiltration in some places than in others; (*b*) from a more extreme vascularization (vascular form), and (*c*) from a predominance of the symptoms of irido-chorioiditis.

*Unequally Infiltrated Forms.*—In the typical variety the infiltration, which to the naked eye may appear to be diffuse, is with the aid of a strong convex lens seen to be interspersed with a multitude of more densely affected

points. This unevenness may be pronounced; the cornea may show large, irregular maculæ of rather intense whiteness instead of the diffuse grayish tint which is seen in the more common variety. Some portions of the cornea may even remain comparatively transparent, and the infiltration, though it is always deep, may be only partial. Numerous small, deep infiltrations situated in front of the membrane of Descemet, and in the centre of a rather transparent cornea, have been described under the name of deep, punctate syphilitic keratitis (Mauthner and others). (See also *Different Forms of Deep Keratitis.*) In rare cases, the more intensely infiltrated spots in the centre of the cornea may become ulcerated (Hutchinson, Treacher Collins). Intense infiltrations leave behind very dense and permanent maculæ, known as sclerosis corneæ.

*Vascular Forms.*—In these types the new vessels, which are developed in the cornea more or less in each case, arise from the deep pericorneal branches; they are so small that they are often visible only through a magnifying glass. In the regressive stage they remain for a long period of time permeable, though empty. Any irritation of the eye, however,—such as a clinical examination,—causes the blood to reappear in them (Hirschberg). Their walls remain as a permanent impediment to vision. Sometimes vascularization exists from the beginning, the cornea appearing uniformly red. These forms are very tenacious and leave dense opacities. In the anterior planes of the cornea vessels may be given off by the superficial ciliary branches and elevate a large portion of the corneal epithelium in the shape of an epaulet. Some authors would regard this as a scrofulous variety of deep keratitis.

*Irido-Chorioidal Forms.*—It has already been stated that there is always iritis, or at least an intense congestion of the iris. In most, if not in all, cases posterior synechiæ, which may become complete and expose the eye to (secondary) glaucoma, are produced. In almost every instance there seem to be deposits on the posterior surface of the cornea. Very often, especially when the affection is one of long standing, evident phenomena of cyclitis and chorioiditis are observed. Chief among these may be mentioned the slaty shade of the sclerotic around the cornea, which is so characteristic of chronic cyclitis. This is due to impregnation of the sclerotic by the pigment of the ciliary body. Sometimes the sclerotic is infiltrated quite as much as it is in scleritis. Subsequently ciliary or intercalary staphylomata may supervene when, in consequence of posterior synechiæ, the eye has become glaucomatous. The corneal inflammation may be so hidden that the condition is spoken of as one of irido-chorioiditis.

During the course of the disease, the ophthalmoscope reveals chorioidal affections, especially about the ora serrata. Hirschberg holds that this occurs in most instances of parenchymatous keratitis, while von Hippel is more specific in stating that it is found in sixty per cent. of all cases. Finally, autopsy has always shown the presence of iritis, cyclitis, and chorioiditis or retino-chorioiditis.

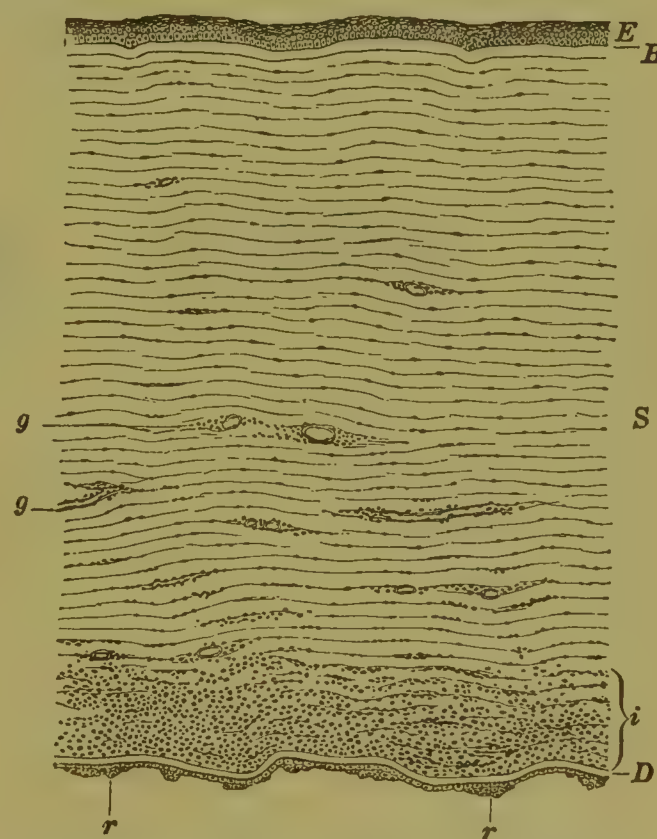
While the fact should be taken into account that, in all cases of this kind that are submitted to autopsy, the disease has been very serious and has destroyed the sight, it remains none the less true that, according to all appearances, parenchymatous keratitis is always accompanied by a greater or less inflammation of the whole uveal tract, especially in its anterior segment. This affection of the uveal tract is by many considered to be the principal feature, the keratitis being, in their opinion, only a complication of the disease.

Michel, in this connection, distinguishes two forms: in the one, the disease commences with the corneal affection; in the other, the corneal affection is absolutely secondary and consequent upon that of the uveal tract.

Those forms in which irido-chorioidal phenomena are predominant are often distinguished by ciliary pains, which become most severe at night when the patient is in bed.

*Pathological Anatomy.*—In the few cases in which the diseased area has been examined anatomically (Fuchs, Brailey, von Hippel) vessels have been found traversing a deep cellular infiltration on the cornea (Fig. 14), which increased in density as far as the membrane of Descemet. There was also an œdematous swelling of the cornea, which was more pronounced in some places than in others. The diffuse initial trouble seemed to be due, in part at least, to this œdema. The cellular infiltrations extended to the irido-corneal angle (the pectinate ligament), the iris, and the ciliary body. Embryonal cells also covered the posterior surface of the membrane of Descemet. In some cases where the disease seemed to be of a scrofulous nature, there have been found areas of infiltration resembling tubercles (Fuchs), even with giant-cells in them (von Hippel and Zimmerman), in the cornea, iris, ciliary body, and chorioid. The last-named author claims also to have detected Koch's bacillus of tuberculosis in the affected portions.

Fig. 14.




Parenchymatous keratitis (Fuchs). *E*, corneal epithelium; *B*, Bowman's membrane; *S*, tissue proper of the cornea, showing a cellular infiltration which commences in the middle layers and increases progressively as far as the membrane of Descemet (*D*), where it is most dense (*i*); *g*, deep vessels in the cornea; the membrane of Descemet is undulated, owing to the unequal swelling of the corneal tissue; *r*, a collection of round cells on the posterior side of the membrane of Descemet.

*Etiology.*—As has been already stated, parenchymatous keratitis has usually a constitutional origin. Moreover, with very few exceptions, it is an ailment of childhood and especially of adolescence.

Since Hutchinson's classical publication on *congenital syphilis* we are aware that this is the cause in most cases. In every instance in which parenchymatous keratitis is observed the parents' history should be carefully inquired into, and search be made for the concomitant symptoms of hereditary syphilis, such as remainders of old fissures, cicatrices at the labial angles or in the mouth and throat, swelling of the lymphatic glands, deformities of the long bones (as at the crest of the tibia), flattening and enlargement of the root of the nose, atrophy of the upper jaw, strongly projecting frontal protuberances, etc. Hutchinson has called attention to two symptoms of hereditary syphilis which frequently accompany parenchymatous keratitis,—namely, a certain degree of deafness (caused by otitis media) and a peculiar conformation of the teeth, "Hutchinson's teeth." (Fig. 15.) Soon after their eruption the upper incisors of the second

FIG. 15.

Hutchinson's teeth, occurring in congenital syphilis.

dentition become narrow at their free margins. The sloping shape becomes more and more pronounced until the teeth are converted into shapeless stumps. Sometimes these teeth and even the entire superior maxillary bone are rudimentary from their origin. The teeth of Hutchinson should not be confounded with those which have a transversely striated surface (rhachitic teeth). As Hutchinson's teeth exist in nearly one-half of the cases of parenchymatous keratitis, their presence is regarded as pathognomonic of congenital syphilis.

The coexistence of these teeth with the defective audition and the interstitial keratitis constitutes the symptomatic "triad" of Hutchinson, which is absolutely characteristic of congenital syphilis. Nevertheless, the teeth may not assume this form in hereditary syphilitic keratitis and they may be only slightly roughened. In such cases it is well to give, if possible, attention to the ophthalmoscopic signs recently enumerated by Antonelli as being present in congenital syphilis,—namely, a certain degree of pallor, sclerosis of the papilla, a massing of pigment on the papillary border, a certain narrowing of the retinal arteries, and a granular state of the retinal pigment. These are not exclusively characteristic of congenital syphilis, but their existence may confirm or establish the diagnosis.

Hutchinson believed congenital syphilis to be the sole cause of parenchymatous keratitis. It is to-day admitted that other conditions may produce it, but that the hereditary syphilitic form constitutes, at least, one-half and perhaps three-quarters of all the cases; Saemisch gives sixty-two per cent., Michel fifty-five per cent., Mauthner eighty per cent., Horner sixty-four per cent., Silex eighty-three per cent., and Panas forty per cent.

According to Parinaud, hereditary syphilitic keratitis betokens that, at the moment of conception, the syphilitic virus in the parents has become somewhat attenuated. While

the preceding pregnancies may have all ended in abortion, this one terminates in the birth of a viable infant.

Acquired syphilis but rarely gives rise to parenchymatous keratitis. The cases cited as such are not all authentic; many are examples of iritis or irido-chorioiditis, which have produced secondarily a deep lesion of the cornea. It is a curious fact that syphilis in the adult very rarely attacks the corneal tissue, though it so frequently affects the iris and even the whole uveal tract.

Scrofula is certainly a cause of typical parenchymatous keratitis; Mackenzie even attributed to it all the cases of this disease. One must admit that in some cases tuberculous deposits have been found in the cornea. Usually the iris, the ciliary body, and the pericorneal zone of the sclerotic were also infiltrated by the tuberculous process at the same time as the cornea. In one case, described by Bongartz, there were tubercles in the sclerotic around the cornea and in the uvea. In this case the cornea showed only a cellular infiltration with vascularization. Some clinical observations of parenchymatous keratitis have been published in which the anterior segment of the eye seems to have been the seat of tubercles. It appears also that tuberculous iritis may develop without the appearance of tubercles; hence their absence does not prove that an iritis complicating an interstitial keratitis is not of a tuberculous nature. Clinical observations also show that tuberculous iritis with parenchymatous keratitis may be cured. Finally, some examples of parenchymatous keratitis, typical at least in the beginning, are occasioned by tubercles, situated either in the cornea, in the sclerotic, or in the iris. This tuberculosis seems to be always of endogenous origin. It would, however, be going too far to regard all the cases of non-syphilitic interstitial keratitis which occur in tuberculous persons as being due to a development of tubercles in the anterior segment of the eye.

In children, small corneal phlyctenules, that are very transient, may give place to a deep, diffuse, tenacious keratitis, which is analogous to the parenchymatous form. During the last two years the writer has, at intervals, treated a young girl who has suffered in both eyes from large and tenacious infiltrations of the corneal limbus, and which were each time complicated by an interstitial keratitis. A portion of the infiltrated area of the limbus introduced into the eye of a rabbit did not cause tuberculosis. Primary and exogenous tuberculosis of the cornea, which have been studied experimentally, particularly by Haensell and Panas, have been but seldom observed in man. A suppurating ulcer developed from eight to fifteen days after the inoculation.

Rheumatism, gout, influenza, impaludism, uterine affections, and contusions of the cornea have been named among the rare causes for parenchymatous keratitis. These cases are, however, often atypical, generally consisting only in deep infiltrations of a part of the cornea, with iritic complications or merely a tendency thereto. Some authors seem to include all these forms under the name of parenchymatous keratitis, but the writer thinks incorrectly. Still others speak of a "parenchymatous keratitis" in those cases of iritis or irido-cyclitis that produce secondary alterations in

the endothelium of the cornea, with a certain degree of œdema. They also even call it by this name when, later on, sclerosis of the deep corneal lamellæ has developed. Thus a physician may seem to have in his practice relatively but a small proportion of cases of parenchymatous keratitis due to hereditary syphilis.

Animals may be attacked by an interstitial keratitis similar to the parenchymatous form which is observed in man. In the febrile disease to which many young dogs are subject may be observed a double parenchymatous keratitis consequent upon the general malady. Hennike has recently described this disorder as it is seen in bears. The particular pathogenesis of parenchymatous keratitis is not by any means satisfactorily known.

According to Panas and Fournier, hereditary syphilitic keratitis should be regarded, not as a localization of the syphilitic virus in the cornea, but as an expression of a cachectic state or dyscrasia of the general system. It will doubtless be admitted that the utility of the iodides, and even of the mercurials, in a disease does not prove that it is of a syphilitic nature.

As the malady is in many cases the expression of a general condition, it is thought that a pathogenic germ,—a microbe or other noxious substance,—has been transported by the blood-current, and, emerging in the pericorneal vessels, penetrated the cornea through the path of the interstitial lacunæ. This is certainly true in regard to cases of keratitis that are due to tuberculosis. Besides a tuberculosis of the uveal tract, there are found, at least in the anterior segment, deposits of tubercle in the sclerotic, around the cornea, and even in the cornea itself.

In Bongartz's case the cornea did not contain tuberculous deposits. The sclerotic around the cornea as well as the uvea was infiltrated by them, but the cornea itself did not show their presence. Von Hippel and Zimmerman have published examples in which the corneal periphery alone contained the tuberculous elements, and yet the cornea was affected throughout. It seems, therefore, that tuberculosis may exert a noxious influence on the tissue adjacent to that which is affected. Special attention should be given to the toxines that are produced by the bacillus of tuberculosis, which may be produced through the cornea, even when only the sclerotic is infected with the germ. It may also be believed, with von Hippel, that the presence of tuberculous tissue in the periphery of the cornea so alters its vascular supply that a keratitis occurs through the failure of nutrition. The resulting lesion may be more or less analogous to the corneal affections that are observed particularly in scleritis, and generally in those affections which involve the sclerotic immediately around the cornea

In this connection should be cited the diffuse form of corneal affection, with œdema and vascularization, which results from section of the greater part of the posterior ciliary vessels (Wagenmann), or from ligation of the venæ vorticosæ (Koster). Wagenmann explains this form of keratitis also by a failure of the nutrition of the cornea. Granting that iritis and chorioiditis are always present in parenchymatous keratitis, it might be supposed that in this disease the corneal affection, in an analogous manner, was produced in consequence of changes in the uveal tract. The experimental keratitis of Wagenmann does not, however, appear to prove very much in this regard. To produce it, the circulation in the major part of the uveal tract must be arrested to such a degree that the eye becomes phthisical, and it is well known that there is a corneal disease in phthisis of the eye, no matter from what cause it may arise. The lesion does not seem to result primarily from the failure of nutrition in the cornea, as its nourishing vessels are not concerned in this experiment.

As a general rule, attacks of irido-cyclitis do not give rise to deep keratitis; they do so, however, (*a*) when they produce notable deposits on the posterior face of the cornea, and (*b*) when they cause grave alterations in the sclerotic around the cornea. Although, in the latter case, the corneal affection only slightly resembles parenchymatous keratitis, it deserves mention in this place. Still, it must not be forgotten that, in the beginning of typical parenchymatous keratitis, alterations in the sclerotic around the cornea are not seen. The only conclusion which can be safely drawn from the experiments of Wagen-

mann is that the nutrition of the corneal tissue depends—more so than is generally admitted—on the intra-ocular circulation, owing to the connection existing between the corneal vessels and those of the uveal tract.

Diseases of the uveal tract may provoke an affection of the cornea in still another manner. It has been demonstrated by Leber that the absence of the corneal endothelium allows the aqueous humor to filter into the cornea and produce œdema and irritation therein. The injection into the anterior chamber of any liquid except the physiological solution of chloride of sodium and the saturated solution of boric acid injures the endothelium and causes irritation of the cornea; it may even give rise to a persistent sclerosis (Nuel). In affections of the iris and in irido-chorioiditis deposits frequently occur on the posterior aspect of the cornea which injure the endothelium and affect the deep corneal planes. (See section on *Lymphangitis of the Eye*, etc.) Even in the depths of the eye toxines may be produced which damage the endothelium and the tissue proper of the cornea. It is not, however, in this manner that the parenchymatous keratitis develops; most often it begins at some part of the corneal periphery, and is not at first widely diffused over the cornea. It is shown elsewhere that the parenchymatous keratitis frequently commences in the corneal tissue before the appearance of deposits on the posterior side of this membrane. In cases of intra-corneal tuberculosis, which are always accompanied by tubercles of the iris, it might be supposed that the posterior portions of the cornea would become infected by the bacilli. Nevertheless, in a case of irido-cyclitis of very long duration, which had in its course produced a deep sclerosis of the cornea, the writer found tuberculous deposits both in the iris and in a fibrinous cellular exudate of the anterior chamber, but the cornea did not contain giant-cells. Proliferation of the endothelium and a diffuse cellular infiltration were also found in the deep corneal planes. In conclusion, the evidence at hand seems to show that tuberculous germs when present in the anterior chamber are not able to penetrate through the membrane of Descemet, even when the endothelium has become altered.

A remarkable example of corneal disease, which is due to the destruction of the endothelium, and which very much resembles parenchymatous keratitis, has been investigated by R. Dubois, who produced it in a dog which was made to inhale chloride of ethyl. The inhalation was continued forty-five minutes, during which period the animal had to be revived two or three times. The cornea became completely opaque and appeared like porcelain throughout. It cleared up in the course of a week's time, the new growth of endothelium being reproduced from the periphery.

*Treatment.*—As this is a constitutional malady, it is clear that its treatment should be general as well as local. The local treatment is, however, quite as important as the general, for it alone can prevent and cure the complications and the consequences of the disease—synechia and corneal scleroses—which are destructive of vision, and against which general treatment is wellnigh impotent.

The first indication in the local treatment is to keep the pupil dilated by atropine, in order that, after cure is obtained, posterior synechia may not diminish the vision or give rise to glaucoma. As the treatment will require a long time, the mydriatic will produce some temporary intoxication, as shown by the dryness of the throat. Dark glasses or a shade should be ordered for the photophobia. A bandage is required only in the extremely rare cases in which the cornea threatens to become ectatic or ulcerated.

This treatment is sufficient in the early stages, when the symptoms of irritation are intense. After one or two weeks' time it is necessary to aid resorption of the exudations and to avoid sclerosis of the cornea; as all the various agencies which favor these purposes are more or less irritating, they should be used with caution. Among them are hot compresses, the pro-

jection of hot-water vapor against the cornea, and the ointment of the yellow oxide of mercury. They should be used for a long period of time, from several months to a year, and in alternation with one another. In syphilitic forms the writer has obtained encouraging results from daily massage of the cornea with mercurial ointment. Injections of bichloride of mercury into the conjunctiva have recently been recommended (Darier, Gallemaerts, and others). After cocainization one or several drops of the corrosive sublimate solution are injected about a centimetre from the corneal margin, the injection being repeated a few days later, when the irritation resulting from the first one has disappeared. Substances thus injected penetrate into the cornea and even into the interior of the eye (Pflüger). In a case in which ciliary injection showed that the second eye was also infected, the corneal affection first appeared in the immediate vicinity of the point of injection, and spread from this place until it became complete. Perhaps these injections should not be made in the early stages of the malady; at a later period, however, when the symptoms of irritation have diminished, their use has resulted in a marked clearing of the cornea. The writer has employed them only in the hereditary-syphilitic form, but from all appearances they would be equally efficacious in the other varieties. The solution of corrosive sublimate thus employed has been of a strength of one part of bichloride of mercury to one thousand of water, but still more concentrated solutions may be used. A 1 to 500 solution of cyanide of mercury seems to produce the same effect, and, moreover, has the advantage of being less irritating. De Wecker has recommended more copious injections, as much as one-half of a Pravaz syringeful.—In cases of obstinate vascularization of the cornea peritomy is to be performed.—The application of leeches to the temple, which seems to be indicated especially in the irido-chorioidal forms, is contraindicated when the general health is bad. —In those cases that are disposed to become chronic, it is scarcely, by any known means whatever, possible to prevent the formation of synechiæ, and in such conditions atropine may produce increased tension. Iridectomy may be beneficial in these glaucomatous forms, especially during the decline of the disease. Repeated puncture of the cornea has been undertaken in the beginning of the affection, but without any very evident success.

*General Treatment.*—The syphilitic forms usually require specific treatment, but it should not be forgotten that the majority of these patients are already cachectic and that the administration of mercurials may be injurious. Their use is, however, to be recommended for patients of relatively robust constitution. Internally, from one to ten milligrammes of bichloride of mercury should be given daily in pill form. To prevent salivation the mouth and especially the teeth should be washed, many times a day, with a solution of the chlorate of potassium. For feeble infants, whether syphilitic or simply scrofulous, a medication that is less depressing is preferable,—iodides, with substantial nourishment, and cod-liver oil. In the treatment of adolescents, hot baths of the entire body, followed by sweating in bed,

will be found beneficial. The disease often appears in the second eye during the course of treatment.

(*b*) **Different Forms of Deep Interstitial Keratitis.**—There are frequently observed cases of deep keratitis, consisting of more or less circumscribed infiltrations, that sometimes increase in size and somewhat resemble those of the parenchymatous varieties. The lingering course and the iridic complications are also the same as are seen in the last-named malady; the two forms, however, can scarcely be mistaken for one another. The reaction is at times intense, though sometimes it is mild. In the former case a surprising clearing of the cornea eventually occurs. In the latter, indelible scleroses of the cornea may remain.

*Etiology.*—After a chill, or sometimes following an attack of articular rheumatism, a rather large and deep infiltration of a dull color appears in the central portion of the cornea. This is attended with rather severe symptoms of irritation (photophobia, lacrymation, ciliary injection, congestion of the iris, and iritis). These infiltrations may thus be caused by contusions of the cornea; they have also been observed in cases of malarial cachexia, influenza, gout, and syphilis. Such a deep infiltration, which is more or less diffused, may start from a small ulcer, especially a phlyctenule of the cornea. They are often found in the new-born, being caused by the application of the forceps. A deep infiltration of the cornea within its whole depth, which leads to corneal sclerosis, accompanies relapsing scleritis. Deep infiltrations which have no tendency to suppurate are usually the result of constitutional diseases.

*Treatment.*—The iris should be examined frequently, and atropine is to be prescribed if necessary. As discutients or resolvents, warm compresses, especially in the rheumatic forms, and hot baths followed by sweating in bed, appear to be useful. If there be photophobia, dark glasses should be worn. The bandage is indicated only in cases in which there are notable abrasions of the surface of the cornea.

(*c*) **Lymphangitis of the Eye.**—This condition has also been described under the names of *descemetitis*, *aqueous capsulitis*, *deep punctate keratitis*, and *serous iritis*. Lymphangitis of the eye, the last of the varieties of keratitis to be described, is situated in the deeper planes of the cornea, but the iris, the ciliary body, and even the chorioid are always attacked. The affection is really a serious inflammation of the entire uveal tract, the cornea being involved only secondarily. The alterations of the cornea are the most striking, and it is for this reason that it has been heretofore ranked among the varieties of keratitis. The corneal lesion here is a striking symptom of a profound malady of the eye. The name deep punctate keratitis leads to confusion with the deep punctate forms of true keratitis, which are situated in front of the membrane of Descemet. The term descemetitis is also incorrect, for the same reason. The name lymphangitis of the eye seems more appropriate (de Wecker).

Concurrently with the symptoms of serous iritis, and perhaps with some

discrete synechiæ, dots appear on the posterior aspect of the cornea. These spots are situated in the lower half of the cornea, and are often grouped in the shape of a triangle, with the base downward. They are frequently very numerous (a hundred or more), and most of them have a diameter of only a fraction of a millimetre. The larger ones, measuring a millimetre, are situated at the bottom, the tiny ones are placed at the top. They often have a brownish, almost rusty tint. On a level with the larger spots the substance of the cornea is dim and its surface is sometimes slightly dull. The deposits on the posterior face of the cornea consist of migratory cells, of fibrin, of various granulations, of some pigment, and of altered endothelial cells (Knies). H. Snellen (junior) has found them to be largely composed of motile bacilli. If this observation be confirmed, it will be of the utmost interest. These deposits, some of which may exist on the crystalline lens, proceed from the depth of the eye, from the iris, from the ciliary body, and from the chorioid (whence also the pigment is derived). They pass through the pupil into the anterior chamber, and under the influence of gravitation and the movements of the eye are precipitated upon the posterior aspect of the cornea. According to the experience of Leber, these foreign particles produce such injury to the endothelium that the aqueous humor may penetrate into the substance of the cornea and cause trouble. The eye may be glaucomatous, possibly from hypersecretion of aqueous humor and occlusion of the irido-corneal angle through exudation. The exudations may be reabsorbed. On a level with the largest spots indelible maculæ sometimes remain.

*Etiology.*—Syphilis, rheumatism, gonorrhœa, and affections arising from the corneal matrix are capable of giving rise to this condition.

*Treatment.*—It is necessary to treat the iritis. Atropine may be contra-indicated, owing to an increase of tension in the eye.

In the rheumatic forms hot baths, followed by free perspiration in bed, have given good results. For the glaucomatous symptoms the local application of pilocarpine is first to be tried, and if this does not succeed corneal puncture or, better still, sclerotomy or, perhaps, even iridectomy should be performed.

The nature of this disease was not understood for a long while, as the posterior synechiæ are usually discrete and few in number. Similar deposits, which are not so numerous and excessively small, are produced in most cases of iritis on the posterior aspect of the cornea (H. Friedenwald, Dunn). These spots are only capable of being seen with a strong convex lens of 20 to 25 D. They generally disappear without leaving behind any trace of their former presence.

(*d*) **Deep Indelible Corneal Affections in Irido-Cyclitis.**—In severe cases of irido-cyclitis of long standing, especially when the sclerotic becomes finally affected, traumatic and other causes sometimes gradually give rise to a dense sclerosis of the cornea. This is situated deeply in the posterior portion of the cornea, and most often starts from the periphery. It is not

the sclerosis itself that demands attention, but the fact that it is associated with a grave malady of the eye, which is more deeply situated and which most often leads to destruction of vision. This deep variety of sclerosis is largely due to an overgrowth of the corneal endothelium, which arranges itself in layers and forms a lamiated membrane that more or less closely resembles the true corneal tissue. Landesberg has recently called attention to these formations, which are so frequently found in eyes that are enucleated on account of traumatism. The deep corneal lamellæ are also affected in an irregular manner. Vessels may develop in the newly-formed tissue. The grave and lasting changes seen in hypopyon are due to alterations that are similar to those which have just been described.

(*e*) **Post-Operative Affections of the Cornea.**—Following operations on the eyes, especially after an extraction of cataract, deep lesions of the cornea may be observed in one of two forms: 1. A diffuse milky thickening of the whole cornea, which leaves a cicatricial cloud. It is a result of the destruction of the endothelium by the presence of abnormal liquids, especially of corrosive sublimate, which have penetrated into the anterior chamber. Even pure water may produce this affection. Only the physiological solution of chloride of sodium and, perhaps, boric acid solutions are inoffensive (Nuel and Cornil). The cornea, divested of its endothelium, becomes infiltrated with aqueous humor. 2. Perpendicular striæ at the point of incision of the cornea. This is an œdema of the cornea produced by the folding over of its edges or only of its deep layers (Nuel, C. Hess).

## C.—SEQUELÆ OR CONSEQUENCES OF KERATITIS.

### A.—CICATRICIAL MACULÆ OF THE CORNEA; KERATALGIA; SCLEROSIS OF THE CORNEA; ARCUS SENILIS.

1. *Corneal Maculæ.*—By the term maculæ of the cornea are designated permanent corneal spots, that are due most commonly to inflammatory processes (ulcers and infiltrations); at other times they are dependent on simple traumatisms. Non-inflammatory and congenital maculæ are rare.

*Maculæ resulting from Inflammation of the Cornea.*—The affected areas may be small or large. The latter sometimes undergo vascularization. When faint, they are spoken of as *maculæ* or *nubeculæ corneæ.* When very distinct, white, and tendinous, they take the name of *leucoma. Sclerosis* of the cornea is also a term that is applied to this condition when the macula is very dense. If there has been perforation, the cicatrix and the iris may become adherent to the macula, especially at the pupillary periphery (adherent leucoma). The anterior chamber is diminished in depth and is more or less obliterated. Adhesion of the iris is often marked by a black spot on the leucoma.

Large adhesions expose the eye to the danger of glaucoma.

The maculæ which result from ulcers of long standing may be flattened, forming facets on the cornea. After extensive destruction of the cornea, the gap may be closed by a thin cicatricial lamella, which generally becomes ectatic. A projecting cicatrix is rarely produced by hypertrophy of the conjunctival tissue or of the epithelium.

Maculæ consecutive to inflammatory processes are usually due to the presence of a cicatricial fibrillary tissue, which has not acquired either the microscopic or the macroscopic appearances of normal corneal tissue. (See *General Pathology.*)

In time, especially when the maculæ are exposed abnormally to the weather and to continuous irritations, they may undergo degeneration and become the seat for the deposition of substances which render them white, tendinous, and porcelaneous. The most common form of degeneration is the hyaline. Fatty degeneration and the deposition of lime-salts have also been noted. These deposits may become deeply diffused in the corneal tissue, or they may be superficial, subepithelial, or even intra-epithelial, when they are more or less circumscribed, giving a bosselated appearance to the corneal surface.

2. *Hyaline degeneration* may affect the epithelium or the substantia propria of the cornea. The substance thus produced is refractive, and arranged as granular deposits of various sizes or as large irregular globules. The hyaline material is produced by a transformation of the albuminoids and is closely allied to the amyloid substance. It differs notably from the latter in not turning brown upon the addition of a mixture of iodide of potassium and sulphuric acid. Both are colored by aniline dyes, such as fuchsin and gentian violet. Sometimes, especially if the tissues have remained for a long time in Müller's fluid, the coloring only takes place after the sections have been left in the staining solution for a considerable period of time.

Fig. 16 is an example of hyaline degeneration occurring in the parenchyma of the cornea, described by Saemisch under the name of *colloid of the cornea.* Somewhat beneath the capsule of Bowman (*E*) is seen a collection of these globules of various sizes (*D*). At *m* the degeneration is progressing. The hyaline deposits are manifest in the lamellæ of the corneal tissue. These are not degenerated cells, as held by von Hippel. By becoming confluent, globules of various sizes may be produced.

When the degeneration affects the epithelium the hyaline material may diffuse itself in the protoplasm of the cell. (See *Bullous Keratitis.*) In such a case irregular granules, analogous to those seen at *m* in Fig. 16, appear in the midst of the protoplasm. Hyaline degeneration occurs frequently in many of the corneal affections, and seems to be especially due to defective nutrition. It is found in filamentary and bullous keratitis, in *keratitis en bandelettes*, in the so-called essential scleroses of the cornea, in arcus senilis, and in pterygium. While not wishing to deny the existence of fatty degeneration of the cornea, the writer would call attention to the great similarity in appearance of the hyaline masses and the fat-globules, and the ease with which the one could be mistaken for the other. The deposition of lime-salts has been proved, but it is well to remember that the hyaline substance itself may become impregnated with lime-salts.

The corneal epithelium covering the cicatrices may undergo hypertrophy and become cornified, recalling to mind the appearance of the epidermis. Sometimes the conjunctival tissues starting from the periphery of the cornea penetrate, as in *keratitis en bandelettes*, between Bowman's membrane and the epithelium. Cicatrices of the cornea, more especially those that have undergone degeneration, are capable of giving rise to ulcers that are very rebellious to treatment and prone to undergo relapses.

Bagius recently described a case in which all parts of the cornea—the physiological and the pathological, the epithelium, the proper substance, and the exudative particles—had undergone this hyaline or colloid degeneration.

Superficial ulcers, especially in young people, may heal if cicatrization goes on rapidly, without leaving any notable maculæ. Surprisingly good results are sometimes seen in the clearing up of large spots that are due to

Fig. 16.


Hyaline degeneration of the parenchyma of the cornea.

gonorrhœal infection in the new-born. A loss of substance which extends as far as one-half of the thickness of the cornea, and especially when it is accompanied by suppuration and perforation, even if caused by traumatism and cured aseptically, will leave indelible maculæ. The same sequelæ almost always result from deep infiltrations that have existed for a considerable period of time. The superficial and the deeper vessels of the cornea, especially after long-continued inflammation, form permanent maculæ.

*Diagnosis of Maculæ with Inflammatory Infiltrations.*—Either of the three pathognomonic symptoms of keratitis—ulcer of the cornea, dulness of the corneal surface, and ciliary injection—may be absent in cases of maculæ. The surface of a macula may be bosselated but not really dull. Maculæ may be vascularized with a small amount of injection of the ciliary trunks, but no capillary pericorneal injection. A much more difficult case is that of ulceration in an old cicatrix. In each case it is necessary to observe the inflammatory areola of œdema surrounding the dense infiltrations, which may for a long time persist around an intense macula resulting from ulceration. This œdema, indeed, may be more or less cellular in character and leave permanently a diffused thickening or a scar around a leucoma. The cicatricial areola is then distinctly limited by an irregular line bordering the transparent part of the cornea; the inflammatory areola gradually disappears in the cornea. This sign marks the distinction between an old nubecula and a recent slight infiltration, especially in an eye in which new or fresh infiltrations (phlyctenules) occur. The surface of the nubecula is not dull. These old, faint maculæ are white; while the recent œdematous ones are of a grayish shade.

*Visual Disturbances.*—Vision is more or less decreased whenever the macula encroaches upon the pupil, although it seldom becomes an obstacle to the reception of light into the eye. On the contrary, light penetrates the globe freely, as in cases of mature cataract. The visual troubles result from the diffusion of light in the eye; each cicatricial particle becomes a refracting body, and the light is scattered, like that which passes into a room through windows made of ground glass.—Again, irregularities of the cornea may complicate a macula: for example, after granular pannus and phlyctenules of long duration the cornea may resemble a cut diamond. From this condition prismatic deviations of the light (irregular astigmatism) and even monocular polyopia may result.—The diffusion is less pronounced in cases in which the maculæ are denser and more opaque. Such a leucomatous macula, covering a part of the pupil, diminishes the vision less than a slight one (nubecula) under the same circumstances.—The inflammatory œdema surrounding a true cicatrix usually disappears. When it has been situated in front of the pupil, its removal may be followed by a marked increase in visual acuity.

If there be a small central macula, the patient may see best in a dim light and also after instillation of atropine, the improvement of vision in both cases being due to pupillary dilatation.

Amblyopia resulting from maculæ of the cornea may produce nystagmus, especially when they have occurred at an early age, as in cases of gonorrhœa of the new-born, and are large enough to preclude useful vision. It is probable that in the absence of any distinct vision, the directing power of the fovea over the ocular movements being absent, normal innervation does not develop in the infant. Perhaps the centre of the retina remains functionally defective, as in congenital cataract. At any rate, nystagmus does not occur when the maculæ are formed in adult life.

The relative frequency of myopia in eyes having corneal maculæ has been remarked by Chauvel. The common occurrence of convergent strabismus under the same circumstances even in emmetropic eyes is well known. Divergent strabismus is also sometimes produced in cases showing high degrees of myopia. The patient must closely approach visual objects in order to augment the size of the retinal images; an emmetrope with maculæ corneæ must make the same efforts of accommodation as the hypermetrope. Convergence and accommodation, which are causes of myopia and convergent strabismus, are exaggerated.

*Treatment.*—In some cases a macula may disappear spontaneously, particularly if it be recent and not too intense. The clearing of all kinds of maculæ is favored by means that are more or less irritating to the eye,—as hot compresses, steam, ointment of the yellow oxide of mercury, accompanied by corneal massage through the eyelids, mercurial ointment, stimulating collyria, laudanum, etc. Electricity has been tried, but without much success. Scraping of the cornea or cutting off the superficial layers with a cataract knife has but little effect except in the rare cases in which the affection is limited to the superficial layers. Peritomy should not be attempted. Perhaps subconjunctival injections of antiseptic solutions merit a renewed trial. Rothmund long ago recommended the use of water under similar conditions.

It has been attempted, after the example of the ancients, to insert in a cornea that is completely leucomatous a button of glass, of celluloid, etc.; the essay, however, resting upon a physiological absurdity.—In our day, renewed efforts have been made to fill up a gap of leucomatous cornea with tissue that has been taken from a transparent human or animal cornea (keratoplasty); the grafts may become attached organically, but the tissue afterwards becomes cloudy, and upon the whole the optical gain amounts to nothing. These attempts, inaugurated by Reisinger, have been repeated by a great number of physicians (see vol. iii. p. 832). Von Hippel, who seems to have pushed these experiments the furthest, proceeds in the following manner. He requires that the anterior chamber shall be present. Only a small central disk is incised, not the whole cornea, as recommended by Reisinger; hence the membrane of Descemet and the deep corneal portions remain, and the aqueous humor does not bathe and infiltrate the transplanted piece or the edges of the wound. By means of a small trephine, von Hippel cuts a disk in the centre of the cornea, and afterwards

removes it with scissors and scalpel. In this space he transplants a circular piece of equal size, which has been taken, through its entire thickness, from the cornea of a chicken. This method is preferred to the one of inserting only a part of the thickness of the cornea of a dog or a rabbit. It is not necessary otherwise to fix the transplanted disk, which adheres if suitable antiseptic precautions have been taken. Both eyes are covered for some days with a bandage, and rest in bed after the operation is insisted upon.

In cases of superficial maculæ the vision is not as likely to be improved as it is by the easier operation of the scraping or cutting of the cornea. Sometimes the wearing of a stenopæic diaphragm, joined to an appropriate convex glass for near vision, may improve the sight.

Iridectomy may accomplish two very useful objects,—the one antiglaucomatous, the other optical. In cases of maculæ with adhesion of the iris glaucoma may supervene from well-known causes. The antiglaucomatic remedy consists in a *large* iridectomy, which is best made above. Eyes of this description should also be carefully watched to guard against the danger of glaucoma. An iridectomy, when made with the object of restoring vision, should, if possible, be *small* and by preference be placed inward. Of late years external iridotomy is preferred to iridectomy. A radiating incision is made in the iris, which has been released from the eye through a corneal wound. The iris is then replaced in the anterior chamber. It is necessary first to ascertain that the retina acts normally, for it may be detached or may be absolutely glaucomatous. This is done by examining the eye in the same manner as in cases of cataract. The optical gain is usually less than the appearance of the cornea would seem to indicate. The reason for this is that the cornea appears more transparent than it really is, when it is viewed over the shining bottom of the iris, or pus may have become infiltrated either behind the crystalline lens or near the ora serrata in the vitreous body. (See *Hypopyon.*) Iridodesis (Critchett) and iridencleisis (see *Operations*) are no longer performed, on account of the lasting irritation and even sympathetic conditions to which they sometimes give rise.

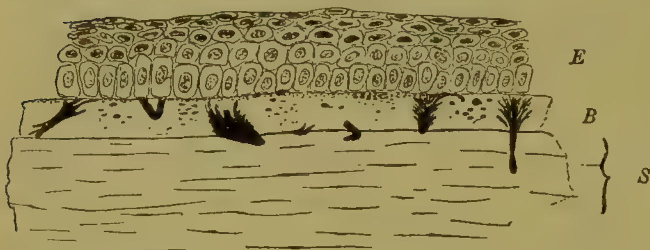
Tattooing of the cornea (see vol. iii. p. 833) is done chiefly for its cosmetic effect (de Wecker). Vision is but rarely improved; when tattooing does accomplish this optical purpose, it is brought about in the same way as through the stenopæic slit. After cocainization of the eye, the Indian ink may be inserted with the aid either of a bundle of needles (Taylor) or by a single point pushed in superficially and obliquely (de Wecker). The Indian ink and needles must be rendered aseptic by heat. Adhesions of the iris expose the eye to the danger of infection, or at least to an iridocyclitis. It is usually necessary to repeat the tattooing on several occasions, a certain period of time being allowed to elapse between the applications.

3. *Keratalgia.*—Small cicatrices of the cornea, which usually result from traumatic erosions, and not from ulcers, sometimes remain painful for

months or more. Ciliary pains, with lacrymation and ciliary injection, occur at intervals, often in the morning, and last for an hour or two. These attacks are neuralgic in character, in the height of which the epithelium of the cicatrix may appear dull. Internal remedies, such as quinine and antipyrine, have but little effect other than of a palliative nature. Massage of the cornea with the yellow oxide of mercury ointment often leads to a cure (Gillet de Grandmont). In an obstinate case I would excise the cicatrix, which is probably strangling or pressing upon some nerve of the cornea.

4. Dense *maculæ of incrustations of lead* are sometimes produced from applications to the eye of collyria made with a solution of acetate of lead. Such particles may be eliminated from the bottom of an ulcer, but if, in the mean time, the epithelium covers them, they will become permanently embedded in that situation. Incrustation almost always takes place in Bowman's membrane (Fig. 17), and can be removed with that membrane. Collyria of acetate of lead should never be used in ulcerative keratitis.

Fig. 17.




Precipitation of lead on the cornea resulting from the employment in a corneal erosion of a collyrium composed of a solution of acetate of lead: *E*, epithelium; *B*, Bowman's membrane; *S*, parenchyma of the cornea. The lead is deposited almost exclusively in a granular form in Bowman's membrane, and in thicker trails in its nervous canals.

5. *Essential Sclerosis of the Cornea.*—In the preceding paragraphs we have spoken particularly of corneal opacities which follow a loss of substance. Corneal maculæ originating from whatever cause are often described under the name of "corneal sclerosis." Here should be noted the maculæ that are consecutive to pannus and to parenchymatous keratitis, also those which result from vegetations of the corneal endothelium, and finally opacities that originate in irido-cyclitis and in scleritis. Under the name of corneal sclerosis *par excellence* is described an affection in which the cornea becomes porcelain-like, slightly vascular, and irregular as to its surface. The affection extends from the periphery towards the centre, as if it were being encroached upon by the sclerotic. This opacity is a consequence of certain forms of scleritis, and lessens but little in the course of time. In one of the published cases it was due to fatty degeneration of the tissue of the cornea (Baumgarten); and in another to hyaline degeneration (A. Schiele). In a word, it results from regressive metamorphosis of the tissue of the cornea and that of the cicatrix, and occurs in cases of long-continued faulty nutrition thereof. These maculæ, especially all those which degenerate, are not capable of being cleared up, and when they are deep, as they usually are, the practice of corneal abrasion is of no utility.

6. *Ribbon-Shaped Affection of the Cornea or in the Form of Transverse Bands.*—Special mention should be made of a rather frequent corneal dis-

FIG 18.


Primary degeneration of the corneal epithelium in the form of inflammation occurring in band-shaped affection of the cornea. *T*, normal corneal tissue; *D*, Bowman's membrane; *n*, normal epithelium; *h*, hyaline globules in the abnormal conjunctival tissue.

ease which was described by von Graefe under the title of "Bandförmige Keratitis." On a plane with the palpebral slit a grayish or somewhat yellowish, non-vascularized inflammation appears, which extends transversely across the cornea; its surface is finely granular, or rough like granite, and rather dry. The affection generally begins at both extremities of the horizontal diameter, though it sometimes starts in the middle; both extremities, however, remaining transparent, somewhat as in arcus senilis. The eye thus attacked is always more or less irritated. The malady occurs most frequently in patients who have suffered for a long time from irido-cyclitis or glaucoma. It is then called "secondary." There is also a "primary" form, which attacks eyes—either one or both—that do not appear to be otherwise diseased. According to von Graefe, eyes suffering from the primary variety often become glaucomatous and should always be regarded as being predisposed to glaucoma. Nettleship includes acute cardiac affections and renal disorders as causes of this ribbon-shaped affection of the cornea. Nettleship and Goldzieher have found deposits of hyaline globes similar to those previously mentioned as existing in certain corneal cicatrices under the epithelium. For this reason the alteration may be looked upon as rather superficial. It appears that hyaline deposits may later on become the seat of calcareous concretions.

In a rather atypical case of the secondary form, the writer found on the surface globular hyaline masses under the epithelium and hyaline degeneration of the cells composing its middle layer, identical with that which has been described as a primary alteration in bullous keratitis. We have been inclined to view this degeneration as the real cause of the disease. Fig. 18 represents, in our opinion, the primary alterations which occur in cases of corneal inflammation due to band-shaped affection of the cornea. This glaucomatous eye was enucleated because of constant pain. Under the epithelium, in Bowman's membrane, may be seen a peculiar conjunctival tissue, non-vascular, poor in cells, with a clear intercellular substance, somewhat resembling the mucous tissue. It not only insinuates itself under Bowman's membrane and the epithelium, but also penetrates into the latter in the form of cylinders and elevates the surface in small protuberances. Its general clinical appearance recalls slightly that of an epithelioma, but it bears no resemblance to pannus. In Fig. 18, at *h* may be seen some hyaline globules, which, however, were more numerous in other portions of the growth than in that chosen for the illustration. The abnormal conjunctival tissue undergoes hyaline degeneration, and may later on become infiltrated with lime-salts. Subsequently the hyaline formation, primarily subepithelial, seems to be able to invade the superficial layers of the tissue proper of the cornea. This explains the fact that certain authors have described a hyaline degeneration that is situated deeply in the cornea.

As for treatment, nothing can be hoped for a clearing up of the maculæ. If the eye be not amaurotic, scraping of the cornea (see vol. iii. p. 821) may be beneficial, as the reformed tissue is ordinarily more transparent than the macula. In the primary forms atropine must be avoided. Miotics should be prescribed, and if the ocular tension be augmented, iridectomy will be clearly indicated. If vision be wholly lost and the eye is painful, relief may perhaps be given either by enucleating the eye or by reducing it in volume. (See *Staphyloma of the Cornea*.)

7. *Arcus Senilis; Gerontoxon.*—In old age, and earlier in certain families in which it is hereditary, a whitish ring appears on the circumference of the cornea; it is larger above and below, is strongly delineated at its periphery and does not wholly reach to the limbus; towards the centre of the cornea it becomes imperceptible. This arcus senilis, which causes no unfavorable symptoms, is the result of a hyaline degeneration of the substance of the cornea, especially of the superficial planes. It was formerly believed to be due to fatty changes, but this is not the case. The presence of an arcus senilis does not interfere with, or check, the cicatrization of the corneal incision made in the extraction of a cataract.

8. *Congenital Maculæ of the Cornea.*—It is by no means exceedingly rare to find in new-born infants, without other anomalies, maculæ on one or both corneæ. They are ordinarily situated centrally. Now and then there is at the same time an anterior synechia. Frequently, the cornea is unusually small,—microcornea. Corneal staphyloma is sometimes congenital. It is not always known whether these anomalies are due to vicious embryonic development, or to an intra-uterine corneal disease. Either cause appears possible.

### B.—STAPHYLOMA OF THE CORNEA; FISTULA OF THE CORNEA.

Staphyloma corneæ is an ectatic cicatrix composed chiefly of the iris and, in varying proportion, of the tissue of the cornea, or of cicatricial tissue which has replaced that of the cornea. The iris usually forms the largest part of a staphyloma. It is always consecutive to a perforation with more or less extensive destruction of the cornea. Staphylomata are divided into partial, which occupy only a portion of the area of the cornea, and total, which includes the whole area of the cornea as far as the sclerotic. Partial staphylomata have a tendency to develop into the latter form.

1. *Total Staphyloma.*—A total staphyloma is most frequently consecutive to an almost entire destruction of the cornea. It is formed at first by the iris, and is then reinforced at the periphery by a remnant of the corneal tissue. The iris is transformed into a fleshy membrane, and afterwards into a cicatrix more or less thin, covered in front by epithelium. This cicatrix bulges more and more, being pushed forward by the aqueous humor; it is first grayish, then white or slate-colored, this being due to the pigment of the iris, as seen by transmitted light. The ectasis may be globular and resemble a grape. Its surface may be bossed, and strengthened in parts by denser cicatricial ridges. If the staphyloma was at first partial, being centrally located, it may become conical, its periphery being reinforced by thicker vestiges of the cornea. The surface of the staphyloma is traversed by vessels. The iris adheres to the remnant of the cornea as far as the periphery, there being no longer any anterior chamber. (Fig. 19.) The eye is always more or less hard. As a sequel, the outer layer of the staphyloma frequently thickens, chiefly in the centre; at other times the latter is thinner.

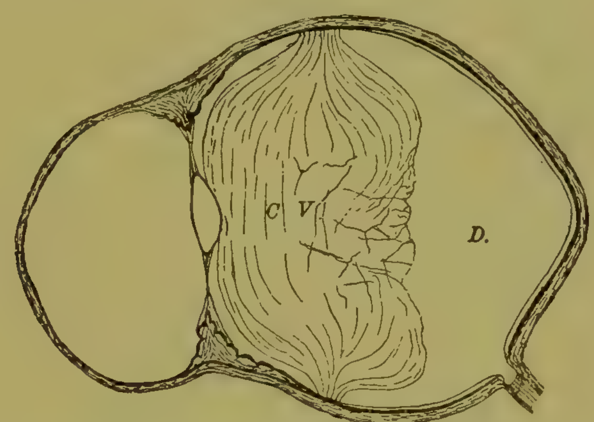
The ectasis becomes larger, is always accompanied by irritation, dragging of the tissues, ciliary pains, etc. A hernia appears between the lids, which no longer cover it. Then the epithelium may thicken, and more or less resemble epidermis. The wall may later undergo all the degenerations described when treating of corneal opacities. Thus exposed to attacks of every sort, the staphyloma may become ulcerated and perforated. The opening closes, and distention reappears. A panophthalmitis or a hemorrhage after perforation may lead to phthisis bulbi. The sclerotic may take part in an ectasis, the eye becoming buphthalmic, especially in children. (Fig. 20.)

Vision is always abolished, and finally the sensibility of the retina becomes completely extinct, most frequently through glaucoma. A glaucomatous excavation of the optic nerve is often produced. The vitreous humor becomes softened and reabsorbed. The chorioid, the retina, and the ciliary body undergo atrophy. The crystalline lens may have been torn loose and removed at the time of the perforation ; if not, it becomes cataractous. It may also be dislocated, but even in cases of buphthalmia it is sometimes kept in place by the greatly elongated suspensory ligament. (Fig. 20.)

FIG. 19.

Corneal staphyloma of a more or less conical shape. The suspensory ligament is stretched or expanded. The crystalline lens is cataractous.

FIG. 20.




Buphthalmos in consequence of a corneal ulcer acquired in infancy. All the diameters of the eye are increased. There is adhesion of the iris to the cornea throughout its entire extent. The suspensory ligament of the crystalline lens is stretched. The vitreous humor (*C V*) is separated from the fundus by a liquid exudate (*D*), but is still adherent to the ora serrata. There is a glaucomatous excavation of the optic nerve.

2. *Partial Staphyloma.*—Partial staphyloma is brought about in the same manner as total staphyloma. It results, however, from a more circumscribed hernia of the iris. Simultaneously with the ectasis of the cicatrix the surrounding cornea yields to the intra-ocular pressure and forms a cone, the apex of which, centric or eccentric, is the cicatrix. The iris is still to be seen through the cornea, and there may also remain a little of the anterior chamber. A certain degree of vision exists in cases in which the pupil is not entirely involved or covered by the cicatrix of the

cornea, or it may be afterwards lost by glaucoma. The ectasis often increases and becomes general and more conical. (See also *Fistula of the Cornea.*)

A perforation of the cornea, most frequently caused by ulceration and suppuration, is occasionally the cause of staphyloma. It is most to be feared in cases of large ulcer, the thinned borders of which cannot resist the intra-ocular pressure. Sudden efforts of every kind are factors which prevent a cicatrix from becoming firm, and even cause one which was at first flattened to become ectatic.

A large thinned cicatrix yields to the normal pressure of the eye. The mechanical conditions (extensive anterior adhesion of the iris), however, are such that from the first the eye is glaucomatous. If, on account of the easy filtration of aqueous humor through the thin cicatrix, the tension is not at once exaggerated, it will soon become so. While a portion of the anterior chamber may still remain in the beginning, it generally becomes at last completely obliterated. A staphylomatous eye is always a glaucomatous one.

*Treatment.*—Every prophylactic precaution should at once be taken. This includes relative rest in case of prolapse of the iris, and prohibition of all fatiguing corporeal labor, even for a long period of time after cicatrization has occurred. An eye in which there is a large cicatrix of the cornea, with adhesion of the iris, must be watched, and, if possible, an iridectomy should be made as soon as the tension increases, or even before.

A partial staphyloma can be cured with the preservation of a moderate amount of vision, but the transparent parts of the cornea have lost their curvature and are always more or less dull. While eserine may be useful, atropine must certainly be avoided. There should be no delay in making a large iridectomy combined with excision of an elongated fusiform piece of the cicatrix, if this be of considerable size. The latter is usually very thin, and in reuniting the edges of the wound greater thickness should be obtained. Transfixion is made with a cataract knife, and excision is then performed by means of forceps and scissors. In somewhat pronounced cases the excision may be followed by corneal suture (Kenneth Scott). As this, however, deforms the cornea, the conjunctival suture is coming to be preferred. That operation is performed as follows: From the conjunctiva behind the wound are taken two flaps, the borders of the ulcer having been deprived of their epithelium by scraping; these flaps are slid across the cornea and sutured. The conjunctiva thus adheres over the wound and strengthens the cicatrix, which shrinks and atrophies on a level with the portion of the cornea that has been covered by epithelium. Further treatment consists in rest and the wearing of a compression bandage for a month longer. These excisions should more frequently than they are be combined with an iridectomy, which is made as large as possible.

Total staphyloma requires a more energetic intervention to relieve the pain and to remove an awkward ectasis. A simple incision is of no use

whatever. If the staphyloma be not excessive, it may be treated by cutting a piece from the side, as in case of partial staphyloma. Staphylomata of good size require a larger incision, including the entire circumference of the cornea and even extending into the sclerotic. As loss of the media of the eye, hemorrhages, and purulent infection may result from the larger wound, its lips must be reunited with sutures. For that purpose Critchett's operation has for a long time been much in vogue. Three large needles, threaded, are passed vertically through the eyeball, half a centimetre from the edge of the cornea, and are left there to support the ball. Then, with a cataract-knife passed in front of the needles, a large horizontal piece, the extremities of which are in the sclerotic, is cut out. Forceps and scissors are used to assist in cutting away the piece as far as the sclerotic edge. This done, the threaded needles are passed wholly through, and the threads are tightened. The result is a horizontal cicatrix with two projecting extremities, which may prevent the wearing of an artificial eye. Therefore preference is now given to excision of the staphyloma with the purse suture of the conjunctiva, either according to the method of Knapp or in accordance with that of de Wecker. (See *Operations*, vol. iii. p. 828.) The two operations are still, however, sufficiently complicated, and the writer often contents himself by operating in the following manner: Around the cornea a thread is stitched well into the conjunctiva. The staphyloma is then excised, and the thread tightened. The operation is easier and gives a regular stump, well adapted to receive an artificial eye.

The evisceration of the eye, or enucleation of its contents, by means of a scoop, after excision of the cornea (Graefe, Bunge), gives too small a sclerotic stump and has no cosmetic advantage over the enucleation of the eyeball. Healing takes place but slowly. In some cases the stump shrinks to a small size and, above all, the liability to sympathetic ophthalmia seems to be greater after this operation than after enucleation. Various attempts are nowadays made to preserve a larger stump, which will give more motility to an artificial eye. Besides the operation of evisceration, already mentioned, it has been essayed (Mules et al.) to enclose within the sclerotic spherical bodies of glass or bone (see *Operations*), also a blood-clot (Venneman). This practice has not met with general favor; healing is always delayed, and the presence of a foreign body enclosed in such a manner does not seem to be devoid of danger.

Enucleation of the eye should be quite exceptional, and should be reserved for cases of buphthalmos, which present conditions excluding protracted post-operative treatment. At first, a stump causes excursive movements of the artificial eye; afterwards the absence of the eye produces a stagnation of the liquids in the orbit, and painful conjunctivitis.

Tattooing of a staphyloma is useless, and exposes the eye to infection.

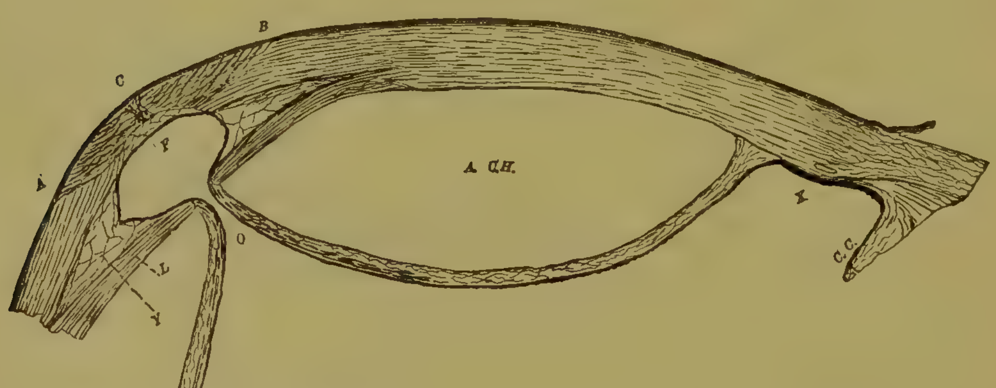
Mention may here be made of *intercalary staphyloma*, which is in reality an ectasis of the cornea and not of the sclerotic. In a glaucomatous eye, often in consequence of irido-cyclitis, a grayish-black ectasis may sometimes appear between the corneal periphery

and the sclerotic. The resulting staphyloma is ordinarily spoken of as a staphyloma of the sclerotic, and which is then differentiated from the ciliary staphyloma of the sclerotic. (See *Ciliary Staphyloma.*) It is noticed that the ciliary vessels do not pass above such an ectasis, but that they spread over a ciliary staphyloma. In reality, this form of staphyloma, which is called intercalary because it seems to develop between the ciliary body and the periphery of the iris, is an ectasis on a level with an irido-corneal glaucomatous peripheral synechia. (See Fig. 21, *X*.) It is from adhesion of the iris to the cornea that the latter becomes more or less softened, so that it here yields to the increased tension. There is, therefore, an ectasis of the corneal periphery, with the neighboring portion of the sclerotic, on a plane with the canal of Schlemm. The ectasis is covered by the iris, which is always atrophied at this plane; it is not the result of an ulcer, but of glaucoma, either primary or secondary.

*Congenital Corneal Staphyloma.*—In the new-born is sometimes seen a corneal ectasis, which is covered on its posterior aspect with atrophied iris. In an eye of this kind which is in our possession the lesion resembles a corneal staphyloma following an ulcer; the crystalline lens is in an embryonic stage, its fibres appearing like large cells. The ciliary muscle scarcely exists. There is no glaucomatous excavation of the papilla, and the retina and the chorioid are atrophied towards the equator of the eye. There is a rudimentary hyaloid artery, and the ciliary processes—properly speaking, folds of the retina—reach to a point behind the crystalline lens; there is also a pyramidal cataract.

3. *Fistula of the Cornea, or, rather, Fistulous Staphyloma.*—A perforation of the cornea may for a week or more fail to close without deserving the name of corneal fistula. This name is properly applied to a perforation of the cornea which is hindered from closing by an epithelial covering.

Fig. 21.




Fistula of the cornea: *C*, spot where the fistula breaks through; *A*, *B*, limits of the cicatricial tissue of the cornea; *F*, intra-corneal gap lined with iris-pigment; *Y*, dehiscence or split in the cornea; *L*, posterior corneal plate, drawn backward and inward by the sphincter of the iris (plainly visible); the iris, or rather its sphincter, adheres to the extremity of this corneal plate; *O*, opening through which the fistulous gap of the cornea communicates with the posterior chamber; *A. C.H.*, anterior chamber; *C. C.*, ciliary body; *X*, anterior and peripheral glaucoma, producing synechiæ of the iris.

Such cases in reality never occur. Czermak observed that the simulated fistula is only periodic, and that it consists of cicatricial tissue in which the sphincter of the pupil is so involved that firm cicatrization is prevented. An anatomical examination of two eyes of this kind confirms Czermak's views.

After central perforation, there is always danger of a central cicatrix of the cornea, with partial or total adhesion of the pupillary sphincter. The anterior chamber still exists to a great extent, at least in the early stages. The cicatrix, which appears as a black spot on the iris, becomes

ectatic. The cornea is thickened and infiltrated, and there develops a small, faint, conical staphyloma, of which the cicatrix is the centre. Later, the cicatrix breaks down, the staphyloma gives way, the opening closes, and the staphyloma is reproduced. This may be repeated a number of times, until, finally, the eye is completely lost by glaucoma.

In a case of this kind the writer found, anatomically, at the location of the cicatrix an excavation of the cornea lined with pigment of the iris. (Fig. 21, *F*.) A vestige of the latter had been taken into the cicatrix. As shown in the illustration, the sphincter of the pupil adheres to a posterior lamina of the cornea, and draws it backward and inward to such a degree as to detach it from the rest of the cornea, and to narrow the opening of the excavation.

*Treatment.*—Where there is peripheral secondary anterior synechia of the iris, it will be necessary to attempt an iridectomy, and especially a sclerotomy. One may even have to excise or to incise the small central cicatrix, for the sake of obtaining a more solid reunion of the edges of the fistula. As in the case of partial staphyloma, the conjunctival suture is to be recommended very highly. Simply cauterizing the cicatrix has not been attended with favorable results.

### C.—MALFORMATIONS OF THE CORNEA: KERATECTASIA; KERATOGLOBUS; KERATOCONUS.

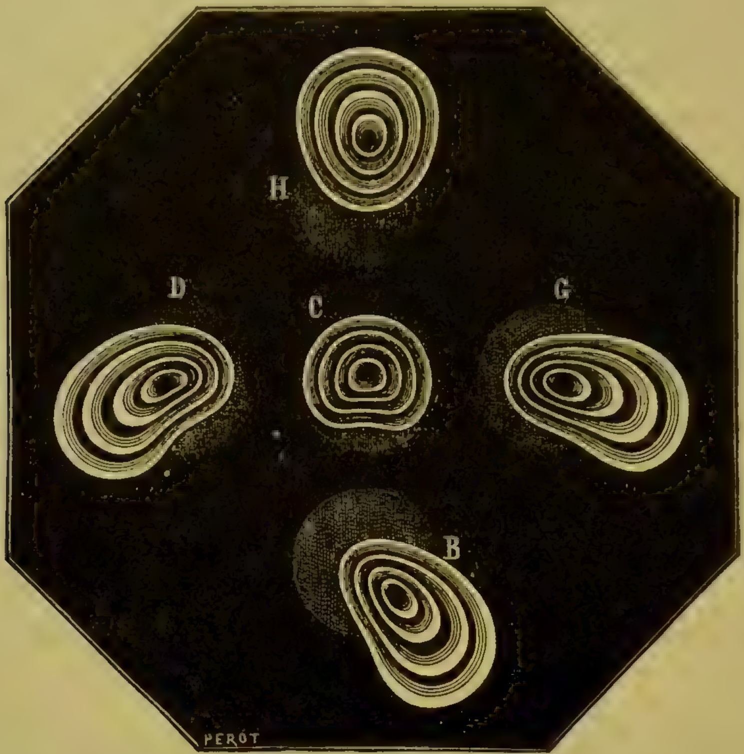
(*a*) *Keratectasia* is an ectasis of the cornea alone, the iris not being involved in it, as it is in staphyloma. A *partial* keratectasia may result from an ulcer which after cicatrization leaves a thin cicatrix; this is rare, however. A keratocele is from a certain point of view a keratectasia. A cornea extensively infiltrated, as in granular pannus or parenchymatous keratitis, may become ectatic throughout. The curvature of the centre of the cornea is then increased, and from this increase an appreciable degree of myopia may result, correction of which is useless owing to an impairment of corneal transparency by the ectasia.

(*b*) *Keratoglobus* is a result not of keratitis but of infantile glaucoma (Dufour). The infantile cornea yields and becomes distended. It is ectatic all over, thin and transparent, with augmentation of its radius of curvature. The anterior chamber is deepened. Vision is diminished or even abolished by the glaucoma, and there is often an excavation of the optic nerve. The eye is sometimes enlarged in all its diameters (hydrophthalmos). (See *Glaucoma*.)

(*c*) *Keratoconus.*—From some unknown cause, the centre of the cornea of a young person may grow thin and be pushed forward. The cornea becomes conical, its periphery flattens, and its centre increases in curvature, but remains transparent. Vision is impaired, and in extreme cases may even be nearly destroyed. (Fig. 22.) In the beginning the patient complains mainly of symptoms of myopia. As the cone enlarges the sight is so much interfered with that it is no longer improved by spherical glasses, and, if

irregular astigmatism supervenes, it falls to a point at which reading becomes impossible. Through the ophthalmoscope the fundus of the eye appears deformed. When illumined from a distance by the mirror, the pupillary field contains a dark ring which surrounds the centre of the cornea.

FIG. 23.



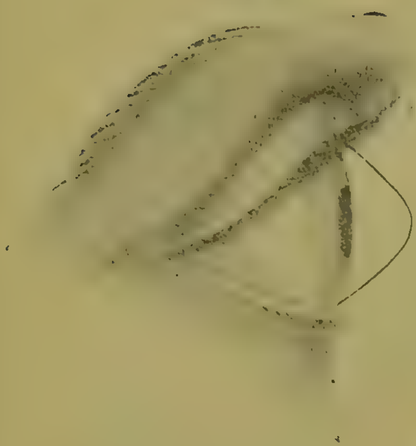

Keratoscopic images in a case of keratoconus

Skiascopy shows a stronger myopia in the centre than in the periphery. Keratoscopic images, small and even regular in the centre of the cornea, radiate to the periphery. (Fig. 23.)

In the beginning of the disease the deformity of the cornea is rather difficult to recognize. When only one eye is affected the observer would naturally think that he was dealing with a case of myopia combined with a high degree of astigmatism. The ectasis, however, becomes more and more pronounced. In a progressing case it may be plainly seen when the eye is viewed in profile. (Fig. 22.) An early diagnosis may be made, however, by observing with the ophthalmoscope the circle of obscuration around the centre of the cornea (Bowman).

Although the cornea appears transparent, there is often found by focal illumination a slight turbidity, which becomes more manifest as the disease advances. The corneal centre is distinctly thinned and is easily depressed by a probe. Intra-ocular tension is normal or slightly diminished. The eye itself is not irritated. In most cases both eyes are attacked, the second one considerably later, sometimes a year or more after the first. In the beginning the ectasis is quite small, but it slowly and gradually augments during several years' time, and finally becomes stationary, when the vision is very much impaired.

Fig. 22.



Profile view of keratoconus.

Fig. 24.


Primary epithelioma of the cornea. *G*, crystalline lens; *I*, iris; *S*, sclerotic; *Cc*, ciliary body; *a*, point where the neoplasm penetrates into the true corneal tissue.

The cause giving rise to this condition is unknown. The affection appears most frequently between the ages of fifteen and thirty years. A feeble constitution has been noted in many of the patients that have been affected. Bowman found keratoconus in several members of the same family. Women are more liable to the disease than men.

Spontaneous rupture of the thinned cornea never occurs. According to Bowman, an abnormal transudation of the aqueous humor takes place through the thinned cornea, so that the balance between the resistance of the cornea and the intra-ocular pressure is re-established. This explains not only the arrest of the ectasis but also the cause for the hypotonus or decreased tension.

The method of production of keratoconus is still unknown. On anatomical examination, Hulke found the membrane of Descemet intact in the centre, and an accumulation of lymphoid cells in the superficial layers under Bowman's membrane. Brailey describes intra-epithelial lacunæ as existing in the extirpated central rings. The stroma was infiltrated with elongated cells. Rampoldi observed the same cellular infiltration of the stroma, and, in addition, found drops of myelin in the epithelium, associated with the absence of the membranes of Bowman and Descemet. It is highly probable that the greater part of these alterations are the result, and not the cause, of the ectasis. The cellular infiltration particularly, which shows itself in the central lesion of the cornea, evidently does not exist in the beginning, and even the thinning of the corneal membrane, apparently the cause of the ectasis, seems partly to be one of its effects. In the experiments of His a central keratectasia was produced by incising the membrane of Descemet with an instrument introduced into the anterior chamber. Elschnig, who has recently taken up these experiments again, compares a keratoconus to an aneurism in which the primary lesion is a lacuna in the elastic tunic of the artery. In his case there was originally a lacuna or an attenuation in the membrane of Descemet. Before him Tweedy supposed keratoconus to result from an arrest of development of the centre of the cornea.

In the treatment of conical cornea efforts have been made to neutralize the irregular refraction by optical measures or to give to the cornea its normal curvature.

*Optical Treatment.*—In the early stages, considerable improvement of vision may be obtained by the aid of appropriate concave glasses, combined, if need be, with cylindrical lenses. This correction, however, does not give satisfactory results in advanced cases. Glasses with hyperbolic curve have been recommended by Raehlmann, but they are not of much value. The abnormal curvature of the cornea is not spherical, nor in reality conical, but it is hyperbolic.

A long time ago Sir John Herschel proposed to correct the refraction by placing over the cornea a transparent shell or cup,—a "contact glass." The suggestion has recently been repeated by Fick and Kalt. Sulzer improved the cup by cutting the glass. When the introduction of air-bubbles between the cornea and the shell is avoided, a notable improvement of vision is obtained. Unfortunately, the contact glass is a foreign body which is not long tolerated.

Favorable effects have always been obtained by the employment of a stenopæic slit, either alone or combined with correcting lenses. G. Mackay has recommended an opaque diaphragm pierced with a series of small openings. H. Snellen has recently, by means of a special variety of the steno-

pæic slit, increased the vision tenfold. In this form, which had already been recommended by Hensen, the horizontal slit, passing from left to right, narrows almost to a point at the centre of the opaque disk. Words viewed through the middle of the slit are distinctly seen; the others, appearing shaded or blended, are successively brought clearly into the line of sight by movements of the head.

*Operative Treatment.*—Sometimes an iridectomy is performed (Tyrrel), which is done for the purpose of admitting light to the eye through the peripheral portions of the cornea. In such a case the retinal image is less diffused. Iridodesis, which at the same time suppresses or displaces the old pupil, would be preferable were it not for the inconvenience of binding in or enclosing the iris. The writer would prefer external iridotomy to iridectomy or iridodesis. Tattooing of the cornea (Grandclément), which produces a central leucoma, seems to be no longer recommended, except as an adjuvant to some operative procedure to be performed afterwards.

Numerous efforts have been made to strengthen the centre of the cornea, to render it more resistant, and to flatten it. (See Operations, vol. iii. p. 824.) Von Graefe observed that a corneal cicatrix following an ulcer often flattened the membrane, and removed a layer comprising the anterior planes by passing a cataract knife at a tangent through the summit of the cornea. Several days later, he cauterized the wound lightly with nitrate of silver. After separation of the eschar, he pierced the base as it arched forward, and, when necessary, kept the perforation open. The result was a central leucoma more or less flattened. Finally, he made an iridectomy.

A. Critchett and Gayet recommend the application of the galvano-cautery to the summit of the cornea, which it cauterizes without always perforating. Even if perforation should occur, it is not a grave accident, for the aqueous humor which flows out cools the hot iron. The effect is the same as that obtained by the preceding method. It is usually necessary afterwards to make an artificial pupil. Tweedy pushes cauterization so far as to cause perforation, which he believes is essential to the production of a flattened cicatrix, and is without danger. Knapp and R. Williams share in his opinion.

Bader excises an elliptical piece comprising the entire thickness of the membrane from the summit of the cornea, which he punctures with a von Graefe knife and then cuts it off with scissors. Adhesion of the iris, which necessitates an iridectomy, always results. Bowman, instead of excising an elliptical piece, removed a disk by means of a small trephine; in this case also adhesion of the iris was produced. Later, he excised a ring comprising all the tissues except the membrane of Descemet, which, bulging into the lacuna, he pierced in such a way as to maintain for some time, perhaps a month, a corneal fistula. The danger of anterior synechia was thereby lessened.—It is evident that in all these operations rigorous antisepsis and the prolonged wearing of a bandage are necessary. None of these efforts have thus far led to a satisfactory result, and we may repeat to-day what Soel-

berg Wells said in 1873, " All these methods of treatment of conical cornea are still upon their trial, and nothing decisive can as yet be said as to their relative advantages or disadvantages."

Miotics, when used for a sufficient period of time, seem to act favorably in cases of conical cornea, the progress of which they moderate or check by reducing the tension in the anterior chamber. Sometimes the only benefit to vision derived from their employment is due to contraction in the size of the pupil.

Panas claims to have obtained satisfactory and lasting results by combining the employment of pilocarpine at least three or four times a day, and the wearing of a compress bandage for a period of from six to eighteen months As this treatment is not at all injurious, it is well to try it in the beginning. If the patient does not submit, or if the effect is unsatisfactory, cauterization with the galvano-cautery may be resorted to, or even excision after the method of Bader or Bowman made. The operation should be followed by the use of miotics and the prolonged wearing of the compress bandage.

## D.—NEOPLASMS AND TUMORS OF THE CORNEA; CORNEAL LEPROSY.

The cornea is so very seldom the seat of neoplasms and tumors that many authors deny that they ever primarily occur therein. On the other hand, the conjunctival limbus is often the starting-point of tumors which may secondarily invade the cornea to a greater or less extent. The tumors of the limbus, however, whether malignant or benign, congenital or acquired, all have one peculiarity in common, and that is their slight tendency to invade adjacent parts, especially the cornea. They usually form more or less prominent projections, which adhere to the limbus, the cornea, and the sclerotics by a slender pedicle that rarely sends out any deep root either towards the interior of the eye or towards the cornea. While there is reason to believe that this vulnerability of the limbus is due somewhat to the great number of its large lymphatic lacunæ, this does not explain the extreme rarity of neoplasms in the cornea, a membrane in which the nutritive interchanges are quite active and which is constantly exposed to traumatisms and infections from without.

It is not our purpose to treat here of sclero-corneal tumors. Of those which attack the cornea exclusively, epithelioma is the only well-authenticated and fully identified neoplasm, and it is very rare. Under the name of corneal fibroma and corneal sarcoma other growths, which were generally small in volume, pedunculated, and formed fundamentally of different varieties of conjunctival tissue, have been described. The designation "fibroma" appears in some cases to have been applied to them incorrectly. After being extirpated they have seldom reappeared.

1. *Epithelioma of the Cornea.*—We are able to present here a well-authenticated example of corneal epithelioma, which has already been published by W. Snellen.

In an elderly man, who had been to the Dutch Indies, a pterygium, that had reappeared after being removed by operation, had given rise to an epithelioma, which had invaded and considerably thickened the whole of the cornea. (Fig. 24.) Several aggregations of cells overlapping each other like so many onions show that the neoplasm, which was apparently epithelial, was really of a cancerous nature. At *a*, the spot where the neoplasm penetrates into the depth of the cornea, the surface looks as though it were embossed. On that side the conjunctival epithelium is slightly thickened, but not as far as the *cul-de-sac*, and the sclerotic is not invaded at all.

In other words, the epithelioma, which originated in the cornea, has developed almost exclusively in that membrane, and has grown only by increase in breadth, no prominent projection appearing on the surface. Having penetrated the cornea, it extends under Bowman's membrane and the epithelium, which are practically intact. Worthy of notice are the thickening of the cornea and the plaiting of Descemet's membrane, showing, as they do, that the neoplasm has involved the cornea only as far as the growth has extended.

The neoplasm shows no tendency to perforate Descemet's membrane, and seems to meet a serious barrier at the periphery of the cornea, for it spreads in every direction over it. Neither does it enter the sclerotic. It is, therefore, distinctly a corneal neoplasm, although it developed in another pathological process which migrated to the cornea before becoming cancerous.

Steiner recently published an analogous case, although he showed some hesitation in diagnosing it as an epithelioma. We once extirpated a small tumor that had developed in the apex of a pterygium. It was five millimetres long by three in breadth, and protruded one millimetre. Comparison would seem to show that these tumors were both of a cancerous nature. It is worthy of note that Steiner's patient was an old Javanese, and that Snellen's case, as mentioned above, was that of a man who had resided in the Dutch Indies.

Growths diagnosed as corneal epitheliomata have been reported by Stellwag, Galezowski, Colsman, and Treacher Collins.

2. *Corneal Sarcomata and Fibromata.*—In a sixty-year-old man Rumschewitsch found a tumor situated within one millimetre of the limbus corneæ, consisting of fibrillary stroma thickly interspersed with fusiform cells and covered with epithelium, to which he gave the name of primary *sarcoma* of the *cornea.* An analogous case is reported by Pagenstecher. Silex designates as fibroma a projection, eight millimetres wide and four millimetres high, covered with epithelium and containing vessels and a number of small cells, which had developed on an old cicatrix. Benson has had a seventy-two-year-old patient with a glaucomatous eye, who attributed to traumatism a small tumor near the centre of his cornea. An analogous case came under the notice of Story and Scott, but the tumor was congenital.

Other anomalous growths which have been seen on the surface of the cornea by various authors can be placed in the category of sarcomata or in that of fibromata with still less certainty than the above.

To sum up, of all the conjunctival tumors found on the cornea some are excrescences from corneal cicatrices, while others are congenital and might very easily be regarded as abnormally implanted dermoids. None of them are reported to have reappeared after being extirpated.

3. *Corneal Leprosy.*—The cornea is sometimes invaded by leprosy in its general tuberculous form, but never, it would seem, by anæsthetic leprosy. The infection evidently takes place in all cases through the limbus,—that is, towards the conjunctiva,—for it is always on the periphery of the cornea (in its tissue proper) that the tubercles first make their appearance. In most instances the iris is invaded secondarily, next after the cornea. The chorioid and the retina may likewise be involved later. The leprotic focus on the cornea consists of agglomerations of young cells which contain Hansen's bacillus and some vessels. The tubercle grows soft, ulcerates, and exhibits a tendency to perforate the cornea; hence iritis, etc., and loss of the eye by atrophy may supervene. Local treatment consists preferably in the destruction of the nodules by thermo-cautery, followed by the usual antiseptic applications and the bandage. As to general treatment, notably the internal use of chaulmoogra oil (up to three hundred or even four hundred drops per day), the reader is referred to the special works treating thereof.

## SCLERO-CORNEAL TUMORS.

(See Tumors of the Conjunctiva.)

## WOUNDS OF THE CORNEA. FOREIGN BODIES.

(See Wounds and Injuries of the Eyeball.)

# DISEASES OF THE LENS.

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## THE NORMAL POSITION OF THE LENS WITH ITS CAPSULE AND SUSPENSORY LIGAMENTS.

THE normal position of the human lens varies very much at different stages of development. When first formed it fills almost entirely the secondary optic vesicle and its posterior surface lies nearly in contact with the retina, while in eyes which go on to proper development the vitreous and its accompanying blood-vessels insert themselves between the lens and the retina, and at birth we find it lying in the anterior part of the eye, its posterior capsule being embedded in the fossa patellaris of the vitreous humor in close contact with the hyaloid membrane, while its anterior capsule is bathed by the aqueous humor and supported at the margin of the pupil by the circular fibres of the iris, which are separated from it only by a capillary layer of fluid. Near the equator it is everywhere held in place by the delicate fibres which its suspensory ligament (the zone of Zinn) sends into the anterior, equatorial, and posterior capsule (Fig. 1). The zone of Zinn is itself firmly attached to the ciliary processes, ciliary bodies, and anterior part of the retina, while it sends a layer forward which dips down into the recesses between the ciliary processes and runs forward to clothe the posterior surface of the iris. Held in place by such delicate but mobile and yielding surroundings, the lens is normally capable of changing its curvature with the varying tension of its elastic suspensory ligament and capsule, and of retaining accurately its place in the eye in spite of the very considerable

FIG. 1.

Insertion of suspensory ligament into lens capsule. (Topolanski.)

shocks imparted to it by the sudden arrest of motion in movements of the eye, and of transmitted shocks of the body and head in various athletic exercises, such as running, jumping, boxing, etc., and even at times of resisting considerable direct violence to the eyeball. Any decided loosening of its suspensory ligament, especially if accompanied by a fluidity of the anterior part of the vitreous, is betrayed by a slight wabbling motion of the iris concomitant with the excursions of the eyes and a variation in the depth of the anterior chamber. While any displacement of the lens either forward or backward must affect the refraction of the eye and either lengthen or shorten its principal focal distance, still it is usually only when the lens becomes either slightly decentred or rotated on one of its axes that the displacement of the image and the astigmatism thus produced markedly interfere with visual acuity and cause the patient to seek medical advice.

The lens may assume an abnormal position in the eye, either as the result of congenital malformation, of disease, or of external violence. In all such cases there is a luxation or dislocation of the lens, but most writers assign to the congenital displacement the name of misplaced lens (ectopia lentis) and speak of displacement due to other causes as luxation or dislocation of the lens, while Becker reserves the latter term for secondary displacement of the lens (as, for example, that due to the formation of a staphyloma of the cornea).

## CONGENITAL DISPLACEMENTS OF THE LENS—ECTOPIA LENTIS.

In some rare cases of so-called total coloboma of the eye the development of this organ is so completely arrested that the lens never comes forward to occupy its normal position, but remains embedded in the vitreous chamber attached to the intra-ocular end of the optic nerve by the hyaloid artery and the fœtal vessels of the posterior capsule, while anteriorly it is attached to the indrawn iris by remnants of the pupillary membrane. Usually, however, congenital dislocation of the lens is due to a faulty development of the suspensory ligament, the lens being pulled toward the point where this is strongest and best developed, and away from the point where it is weakest or entirely wanting. This faulty development or coloboma of the zone of Zinn is at times accompanied by a corresponding coloboma of the lens, iris, ciliary bodies, chorioid, and optic disk. It may, however, occur without any demonstrable lack of development in these organs, and is often accompanied by a flattening or notch in the equator of the lens at the point of the most faulty development of the zonule. This notch, where it exists, is readily seen with the ophthalmoscope as a break in the otherwise continuously curved, blackish line of the lens circumference, and has been described as coloboma lentis. The congenital displacement of the lens is usually either upward and inward, upward, or upward and outward; rarely or never downward, the weak point in the zonula usually corresponding to the position of the fœtal slit. It is

generally found in both eyes and in a symmetrical direction. It is often for years stationary, but there is a tendency as the patients become older for the displacement to increase. The amount of mobility of the lens varies very greatly. In some cases the zonula is so long as to permit of the dislocation of the lens into the anterior chamber, and this may occur while the capsule and zonula are both intact, as is evidenced by the fact that the stretched fibres of the zonula and the corresponding irregularities in the periphery of the lens can then be readily demonstrated with the magnifying glass aided by the use of oblique light. Becker has reported an interesting case, observed in a child of eight years of age at Arlt's clinic, where this excessive mobility of the lens without rupture of the zonula was seen. Here, as in most cases of congenital dislocation, the lens itself was of much less than the normal size. When the lens falls into the anterior chamber it is sometimes free from all zonular attachment, although still covered by its capsule, the delicate fibres between the two having given way. Slight ectopia often passes unobserved in infancy and early childhood and it is only when at school that the child is found to have defective vision. Careful examination usually reveals an anterior chamber unequal in depth, the most anterior part of the lens pressing a corresponding part of the iris forward, while at the opposite side of the chamber the iris not receiving support from the lens lies at a deeper level, and both iris and lens wabble with the motions of the eyes. Looking at the eyes with the ophthalmoscope, even with undilated pupil, usually confirms the diagnosis by showing at some part of the pupil the curved black line caused by the deflection of the return light from the eye ground by the equator of the lens. So long as the luxated lens still occupies the entire pupillary space the symptoms are usually those of myopia and astigmatism, and where the mobility of the lens becomes slightly greater the amount of these defects will vary with the different positions of the head and eyes. Where a displaced lens only partially occupies the pupil and its edge either bisects it or leaves any considerable portion free, the patient with the unaided eye is much annoyed by double vision, the refraction through the free part of the pupil being in high degree hypermetropic (as in the aphakic eye) while that through the part occupied by the lens is still myopic and astigmatic. Such cases also offer a most instructive ophthalmoscopic picture, and as we approach the eye, using the usual concave mirror, we see through the part of the pupil still occupied by the lens a shadow moving with the mirror, and through the lensless part of the pupil another shadow moving against the mirror, while with the plane reflector these conditions are of course reversed. Where we take a condensing lens and use the inverted image to examine the eye-ground, we often obtain simultaneously two images of the fundus, one through the lens and the other through the lensless portion of the pupillary space. Fig. 2, taken from Streatfeild,[1] gives a view of the

[1] Streatfeild, Royal London Ophthalmic Hospital Reports, vol. vii. p. 394.

double images thus obtained with a dilated pupil. This author informs us that "in slight movements of the patient the retinal vessels in the same pupillary area seem to move in two contrary ways at once." The lenses were slightly cloudy, but after a downward iridectomy with $+1/3\frac{1}{2}$ V.=20/XX. In such conditions the lens is sometimes so mobile that by leaning forward, bending the head downward and rolling the eyes in the same direction, the lens will fall forward into the pupillary space, and thus, causing a temporary myopia, enable the patient to read ordinary print when the book is held downward and close to his chest. When the lens still occupies most of the pupil it is advantageous to give the eye a myopic correction, but where most of the pupil is aphakic, a hypermetropic correction (as if for a patient after a cataract operation) is advisable. In either case the correction so much aggravates the defect existing in the other part of the pupil and renders the retinal image received through it so diffuse and faint, that the patient is much less annoyed by double vision than he is when using the eye without a correcting glass.

FIG. 2.

Double images of eye ground seen by use of concave mirror and condensing lens in an eye with ectopia lentis. (Streatfeild).

Ectopia of the lens is often hereditary and existent in several members of one family. Dixon[1] was the first to call our attention to this fact, but since that time numerous instances have been reported. The most interesting and extensive group of such cases of which I am aware is that reported by Morton,[2] and refers to the same family originally studied by Dixon. The family record traces this defect through five generations. The diagrams which he gives of six of this interesting series of cases are here reproduced (Fig. 3) and show the direction and extent of displacement in various members of the family. The acuity of vision obtained by correcting glasses in each is also given, and it appears that all eyes had more or less myopic elongation of the axis, inasmuch as the glasses giving the best vision in the aphakic part of the pupil varied from + 1/5 to + 1/7 or even + 1/10. All the lenses having been dislocated inward or inward and upward, a divergence of the eyes in distant vision occurred when concave glasses were used, while there was convergence when convex ones were employed. The marked influence of heredity is also shown in several interesting groups of cases.

Bresgen[3] reports an instance where a grandmother with ectopic lenses

[1] Dixon, Royal London Ophthalmic Hospital Reports, vol. i p. 54.

[2] Morton, ibidem, vol. ix. p. 435.

[3] Bresgen, Centralblatt für praktische Augenheilkunde, 1897, S. 104.

had two daughters with the same congenital anomaly, and the six children of one of these daughters were all similarly affected. A son, completing the family of three, was born with good eyes, but his only child (a son) had misplaced lenses. Mules[1] relates the case of a father with ectopic lenses, every one of whose ten children were similarly affected. Tiffany[2] observed a family of nine children, seven of whom were affected with bilateral congenital displacements of the lens. The remaining two had no trace of the defect, but healthy hypermetropic eyes (about 3 D.). Miles[3] reports the case of a mother who had ectopia of both lenses, which subsequently fell into the anterior chambers, from which they were in turn successfully extracted. She had eight children, two of whom had good vision, while the remaining six showed marked ectopia lentis.

FIG. 3.

Six cases of ectopia lentis occurring in one family. (Morton.)

Ectopia of the lens is sometimes accompanied by a corresponding displacement of the pupil (corectopia). An instance is related by Alexander[4] in 1874, and later Jones[5] gives a case of symmetrical dislocation of the lens and pupil in both eyes of an individual whose brother presented only corectopia, the lenses being in normal position.

E. Williams[6] describes corectopia with ectopia in both eyes of a brother and sister. Sauvineau[7] reports a case of corectopia with dislocation of the lens in both eyes of a patient with hereditary syphilis and atrophic patches in the chorioid, and Schaumberg one of corectopia with congenital displacement of a notched lens, while Van Duyse[8] gives two cases of double corectopia in which the pupils were displaced outward, the cataractous and ectopic lenses being drawn inward. Breitbarth, Pufahl, Frickhoeffer, Theobald, Wicherkiewicz, and Lindner have all recorded cases of corectopia

[1] Mules, Ophthalmic Review, 1883, p. 84.

[2] Tiffany, F. B., Transactions of the Ophthalmological Section of the American Medical Association, 1895.

[3] Miles, Annals of Ophthalmology and Otology, July, 1896, p 542.

[4] Alexander, Klinische Monatsblätter für Augenheilkunde, 1874, S. 60.

[5] Jones, Dublin Journal of Medical Science, 1879.

[6] E. Williams, Transactions of the American Ophthalmological Society, 1875, p. 291.

[7] Sauvineau, Annales d'Oculistique, July, 1896, p. 55.

[8] Van Duyse, Archives d'Ophtalmologie, December, 1895.

with ectopia lentis. Damianos[1] has found in literature fifty cases of corectopia with ectopia lentis and records two new cases from Fuch's clinic. Ectopia lentis may also occur with congenital aniridia, and cases are cited by Gouvea, Klein, and Rindfleisch. According to D'Oench[2] ectopia lentis occurs once in about five thousand cases of eye disease, while in eighty per cent. of the cases the lens remains clear.

## LUXATION OF THE LENS (LUXATIO LENTIS).

The so-called spontaneous luxation of the lens is always really a secondary process, consequent upon pathological changes which have previously occurred within the eyeball. These are sometimes so slight and chronic that the displacement of the lens affords the first evidence of their existence, while at other times they follow such marked ocular lesions as anterior synechia, staphyloma of the cornea, intercalary staphyloma, etc. Where the displacement is so slight that the lens still remains in partial contact with the fossa patellaris, it is usual to speak of a subluxation, but, when the lens vacates the pupillary space and comes to lie either in the vitreous or in the anterior chamber, the luxation is said to be complete. Luxation of the lens may occur either while that organ is perfectly transparent or after it has become cataractous. If it be quite clear at the time of its displacement it usually becomes cloudy after a prolonged sojourn either in the vitreous or in the anterior chamber, and in either case, even when enclosed in its capsule, it loses weight and becomes smaller. Even in traumatic dislocation, the lens may retain its transparency for several weeks, and in luxation from disease, although it fall into the anterior chamber, it may remain without marked opacity for a long time. Wecker[3] mentions an instance where the dislocated lens maintained its transparency in the vitreous for eight years, and Recordon[4] one where it remained transparent for four years. C. Jaeger[5] claims to have seen it remain transparent for a period of thirty years.

Any such change of place in the lens necessarily implies either a foregoing relaxation or a rupture of the suspensory ligament, and this is usually preceded by a breaking down and fluidity of the anterior portion of the vitreous. Both these changes are in all probability due to pathological alterations in the blood-vessels of the ciliary processes and consequent alteration in the nutrition of the suspensory ligament and vitreous; but our clinical knowledge of the facts has at present far outreached our limited information as to the underlying pathological processes. Becker[6] has called our attention to the fact that in case of opaque lenses the formation of cap-

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[1] Damianos, N., Beiträge für Augenheilkunde, 1897, Heft xxix. S. 8.

[2] D'Oench, Archives of Ophthalmology, vol. x. p. 93.

[3] De Wecker et Landolt, Traité complet d'Ophthalmologie, tome ii. p. 824.

[4] Recordon, Annales d'Oculistique, 1852, p. 233.

[5] C. Jaeger, Dissertatio inauguralis, 1823.

[6] Becker, Graefe und Saemisch, Handbuch der gesammten Augenheilk., Bd. v. S. 290.

sular cataract may be the exciting cause of the rupture of the zone of Zinn. The newly-formed spindle-cells and effused material of capsular cataract are always on the inside of the capsule, and, as they become older, firmer, and more organized, go on to contract, pulling always in the direction of the chord of an arc, and cause the folding of the capsule which we so often find in such cases, and at times by a continued pull in this same direction cause a separation of the fibres of the zonula from the capsule so that the cataractous lens, still covered by its capsule, but absolutely free from its zonular attachments, lies loose in the fossa patellaris of the vitreous. Beer, Wenzel, and others of the older writers have already called our attention to this fact, stating that cataractous lenses on attempts at extraction will sometimes escape from the eye, still enclosed in their entire capsule and without loss of vitreous, and every experienced eye-surgeon has doubtless occasionally encountered a similar state of affairs when operating for the extraction of over-ripe cataracts. The partial displacement of the lens from the pupillary space in any case of subluxation tends constantly to increase. There is a continuous drag from the yet intact or comparatively sound portions of the zonula, and the greater specific gravity of the lens causes it to move with every movement of the eye, thus tending further to break down and soften the vitreous, so that the lens eventually quits the pupillary space and in time usually subsides in the partially softened vitreous humor. In all cases of spontaneous dislocation, therefore, the lens is eventually dislocated downward, and when in cases of long standing we find it dislocated upward we may be sure that the luxation has been either congenital or traumatic. How easily partially dislocated lenses may at times be entirely dislodged is shown by the case reported by Pufahl,[1] where a partially dislocated lens had drawn the zonula across the pupil and a single stroke of the needle allowed it to sink into the vitreous, the vision rising promptly from counting fingers at two feet to ability to read Jaeger III.

Siebold[2] (senior) gives a most instructive case of spontaneous dislocation of an opaque lens. When he first saw the patient the lens was wabbling with the motions of the eye, and he advised an operation for depression. A year later the patient had a clear and mobile pupil, and exhibited herself with joy as cured without operation. In 1831 Desgranges[3] relates a case of a peasant, fifty-six years of age, where the cataract had existed for nine years, when, while thrashing in a barn, he perceived a sort of shaking in his eye and could once more see with it. Fischer, in his "Lehrbuch" (1846), appears to have seen several such cases, and says, "there are cases in which the cataract spontaneously disappears, either by sinking in the vitreous or by accidental bursting of the capsule," and details a case where the sinking happened while the patient was using a solution of lapis divinus with Sydenham's laudanum, "who subsequently kept the precious recipe as if

[1] Pufahl, Nagel, Jahresb. über die Leist. und Fort. im Geb. der Oph., 1875, S. 410.
[2] Siebold, Himly und Schmidt, Bibliothek, 1802, S. 187.
[3] Desgranges, Compte-rendu des travaux de la Société de méd. de Lyon, 1831, p. 54.

it were gold, and recommended it to all blind people." Sichel,[1] however, was the first to give us an elaborate dissertation on spontaneous sinking of the lens, and in some of the eight cases reported by him the lenses were entirely cataractous, in some partially so, and in others entirely clear. One partial dislocation in a man of eighty was followed by glaucoma, and in one case the dislocation occurred at the age of eighteen in an eye with congenital cataract. His first case was so accurately observed, and followed over so long a period of years, that an abstract of it is instructive. A clerk in the custom-house, aged fifty, always myopic, found his sight beginning to fail. He had previously worn — 1/4 and — 1/3½. The right eye, after becoming so dim as to be useless to him, subsequently presented a narrow field in which he could see at a distance, and in which the vision was improved by a convex glass. This occurred when the left eye had in turn become so dim that he could scarcely see to work, even with his nose almost touching the paper. Still later a similar change occurred in the left eye, and when looking downward a cloud came over his field from above, while when looking up he could see an elliptical disk. Finally, in both eyes these disks entirely disappeared, the opaque lenses having sunk into the vitreous and becoming fixed so that motions of the head did not displace them. The patient six years later was still, by means of cataract glasses, working comfortably at his business as book-keeper. The process of spontaneous sinking took about five years to complete. It may, however, occupy a much shorter period of time. Recordon (l. c.) observed a patient of seventy years of age who, while waiting in the hospital for a cataract operation, was kept abed by rheumatism, and observed one day that while in the recumbent position he could see the upper part of the window-frame, but that this disappeared and his vision clouded when he sat up in bed. In "about eight days" the lens had entirely disappeared in the vitreous.

The immediate results of such spontaneous luxation when the lens has been cataractous are, of course, a source of much satisfaction to the patient. Many such cases are on record, but in most instances the intra-ocular changes which have produced the luxation continue, the vitreous becomes fluid, and the lens, if it rests in the vitreous, not only by its gravity, in the motions of the eye, contributes further to break down its structure, but, acting on the chorioid and ciliary processes as a foreign body, gives rise at times to atrophic processes in these organs, or at others produces attacks of glaucoma. The latter termination is more frequent where the partially dislocated lens presses on the pupillary margin of the iris. The clinical results of these processes are well illustrated by the following cases related by Arlt[2] and by Theobald.[3] The former author saw in a boy of thirteen

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[1] Sichel, Spon. Dislocation und Depression der Linse, Zeitschrift für die gesammte Medicin, Bd. xxx., Hamburg, 1836.

[2] Arlt, Krankheiten des Auges, Bd. ii. S. 278.

[3] Theobald, Transactions of the American Ophthalmological Society, 1892.

years a dislocation of the transparent lenses of both eyes into the anterior chambers. The lens of the right eye was extracted with slight loss of vitreous. Three years later the operated eye remained useful, while its fellow had lost all light-perception. The lens of the unoperated eye had spontaneously fallen back into the vitreous, causing occasional attacks of pain, while a conical staphyloma of the cornea had formed, so prominent as almost to prevent closure of the lids over it. A younger sister of this boy had similar dislocations of transparent lenses into the anterior chambers. The eyes, left to themselves, became glaucomatous and sightless, with atrophy of the iris and dilatation of the pupil. The lenses, which had become opaque, had fallen back into the posterior chamber. In Theobald's case a lad of eighteen years had for ten years been able to shake a dislocated but transparent lens into the anterior chamber, and preferred this adjustment of the eye to any cataract glass which could be supplied him. The lens finally commenced to lose transparency, and later the capsule ruptured, this being accompanied by marked inflammatory action. Lens fragments could be seen floating in the vitreous. A year after this occurrence one of the fragments fell into the anterior chamber and excited inflammation. The eye once more became quiet when, after artificial dilatation of the pupil, the fragment was again shaken back into the vitreous.

Chisholm[1] reports two interesting cases of spontaneous dislocation, in one of which the optic nerve became atrophic in a few months, while in the other vision one year after the accident was still good.

## TRAUMATIC LUXATION OF THE LENS.

Temporary traumatic subluxation of the lens may at times be produced by injuries limited to its suspensory ligament,—as is well shown in the case of Aub, where the eye received a severe blow from a bird-shot, which, however, did not puncture it, but was found lying in the conjunctival sac. On the fourth day after the accident there were ciliary injection, a contracted pupil, and a slight effusion of blood into the anterior chamber. Dilatation of the pupil by atropia showed that the lens was transparent, but that it was tilted forward at its upper outer part. There was metamorphopsia, and a walking-stick held diagonally appeared club-shaped at its upper part while it had its normal appearance when held in a direction at right angles. On the thirteenth day the lens and iris had returned to their normal position, and, although there was slight myopia, nevertheless vision equalled 20/XXX. Perforating wounds of the eyeball may at times be limited to the suspensory ligament and its immediate surroundings, so that a slight change in the form and position of the lens may be the only result of the injury. Thus, Becker[2] details an injury to a boy's eye by a fragment of cap, causing a three-millimetre wound in the sclerotic just below and to the inside of

[1] Chisholm, American Journal of Ophthalmology, April, 1893.
[2] Becker, Graefe und Saemisch, Handbuch der gesammten Augenheilk., Bd. v. S. 293.

the cornea, where after excising the prolapsed iris useful vision returned, although through the coloboma thus produced a well-marked flattening of the equator of the lens could be seen at the part where the suspensory ligament had been injured. Five years afterwards the eye was in the same condition, although the piece of cap still remained in the eyeball. Any injury to the eyeball which presses on the zonula or the lens itself may cause either partial luxation or by interference with the zonula produce unequal curvature of the lens. Berlin[1] reports twenty-one cases of astigmatism after injury which he attributes to hemorrhages in the ciliary body. Schiotz gives a case of lenticular astigmatism produced by the pressure of a cyst of the iris, which disappeared after the removal of the cyst. Injuries limited to some portion of the zonula are, however, exceptional, but it is not unusual to have complete luxation of the lens either into the vitreous or into the anterior chamber as a result of blows upon the eyeball or its vicinity, and the sequelæ are likely to be similar to those following spontaneous dislocation into like positions. Severe blows on the eye often cause a rupture of the ball at the side opposite to the blow, with dislocation of the lens, sometimes causing a complete extrusion from the eyeball; at others the elastic conjunctiva escapes rupture and the lens is driven between the conjunctiva and the sclerotic.

### INCOMPLETE SUBCONJUNCTIVAL DISLOCATION.

In some rare instances the escape is not complete, and the lens lies between the lips of the wound, which may either be gaping or closed tightly enough to cause it to assume a figure-of-eight or hour-glass form. Sichel, E. v. Jaeger, Lederle, Pedebidou, Fano, and Mitvalsky all report cases of such incomplete luxations. In Sichel's case the iris has either been torn out or retroverted, allowing a good view of the lens. (Fig. 4.) Notwithstanding the large rent in the sclerotic, the lens was successfully removed by incision in the conjunctiva a few weeks after the injury, and good vision was obtained by the use of a cataract glass. In Lederle's case a large irido-dialysis permitted a good view of the part of the lens lying within the eyeball.

Fig. 4.

Dislocated lens, caught between the lips of the scleral wound. (Sichel.)

### SUBCONJUNCTIVAL LUXATION.

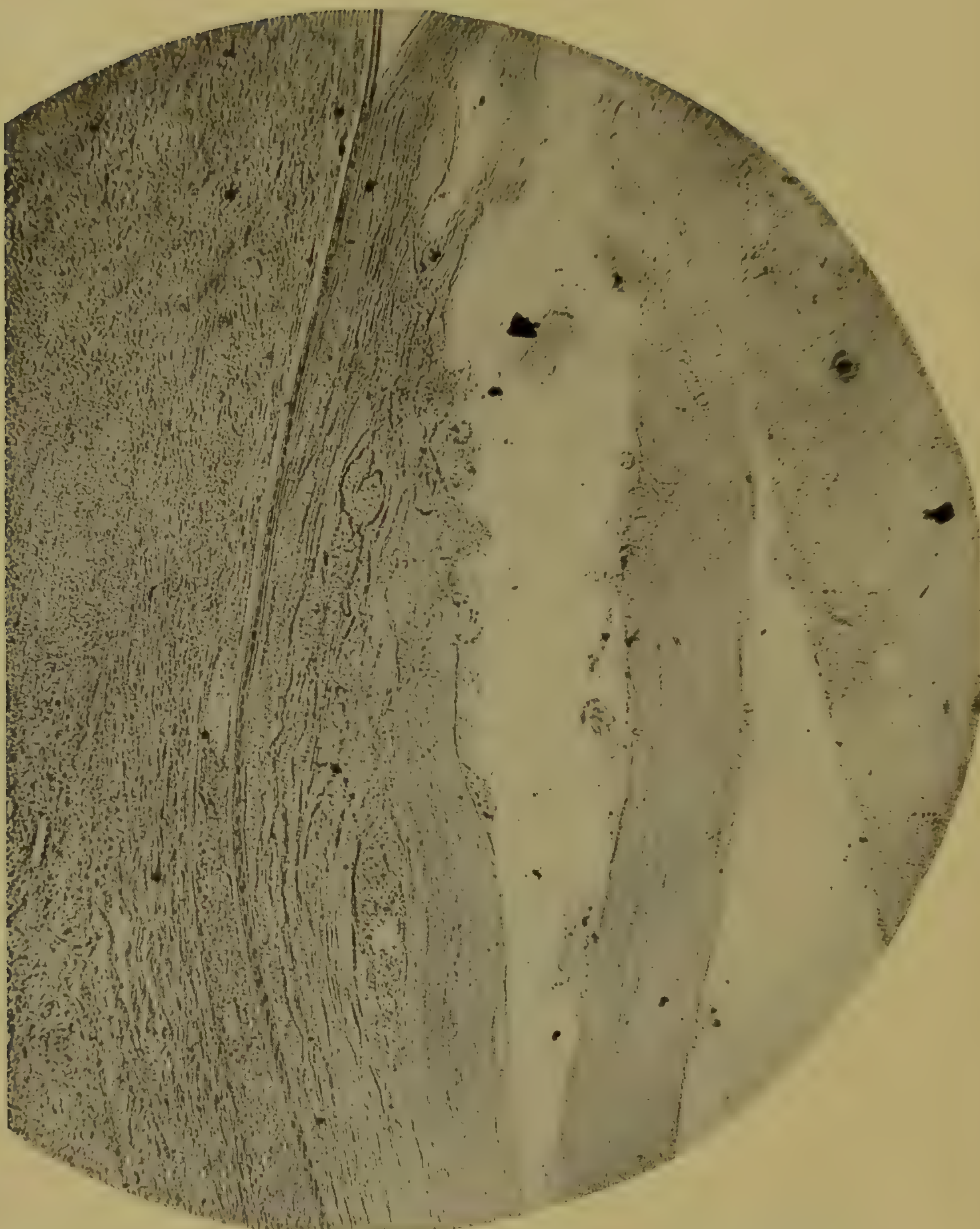
As previously mentioned, a blow may at times burst the retina, chorioid, and sclerotic and drive the lens under the conjunctiva, which by its greater

[1] Berlin, Bericht der Ophthalmologischen Gesellschaft Heidelberg, 1877.

Fig. 5

Subconjunctival dislocation of the lens, with subsequent cataractous degeneration.
(Photographed by Dr. James Wallace.)

Fig. 6.



Subconjunctival dislocation of the lens, with subsequent cataractous degeneration.
(Photographed by Dr. James Wallace.)

elasticity may itself escape rupture. Owing to the protection afforded to the eye by the frontal bone from all blows coming from above, ruptures of the eyeball usually occur at the upper part, being produced by blows coming from below, but Lawson[1] pictures an exceptional case where the rupture of the eyeball occurred at the lower margin of the cornea while the lens lay under the conjunctiva to the inner side of the rent and slightly above it. Treacher Collins[2] also relates a case where the lens was dislocated under the conjunctiva downward and outward. The eye healed and with + 11 D. V. = 20/C. The case was seen eighteen years later, and the eye had not given any trouble. The lens could still be seen as a slightly raised yellow patch at the lower outer part of the globe. Higgins has reported a case of subconjunctival luxation of a cataractous lens, caused by the blow of a fist, where the final visual acuity exceeded that of the fellow-eye which had been successfully operated on in the usual manner. Such injuries, however, by no means always result harmlessly even when vision is retained. Jacobs describes a case where the eyeball was ruptured by the blow of a finger, the lens driven under the conjunctiva, and the iris was either torn out or turned back so completely as to be invisible. Inflammation gradually subsided, but seven weeks later sympathetic iritis developed in the fellow eye and extinguished the sight. The exciting eye with a proper diaphragm and a cataract glass retained a moderate amount of vision.

The accompanying figures are from a case of the author where the subconjunctival dislocation was upward and inward and had been produced by the patient falling against the edge of a stove. The rupture of the sclera was firmly cicatrized, the cicatrix enclosing pigment from the entangled ciliary processes and chorioid. The lens itself was irregularly globular in shape, still enclosed in its capsule, the epithelium of which, although somewhat atrophic, was distincly to be followed as a continuous layer over a considerable part of it. The lens-fibres themselves were everywhere commencing to undergo molecular degeneration. The cataractous changes were more marked near the capsule. Fig. 5 gives a moderately magnified view of the entire region involved, showing the pigmented cicatrix in the sclerotic with the lens in its capsule and lying beneath the conjunctiva. The capsule is adherent to the sclerotic and in places also to the conjunctiva. The latter membrane at one point has undergone considerable inflammatory thickening. The lens is everywhere undergoing cataractous degeneration.

Fig. 6 shows the same preparation, under a higher magnifying power, at a point where the capsule is adherent to the conjunctiva. The capsular epithelium can be distinctly seen in a somewhat shrunken and atrophic condition. There is marked molecular degeneration of the lens-fibres with numerous spindle-shaped chinks between them which are for the most part filled with granular material. Further from the capsule is to be seen a

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[1] Lawson, Injuries of the Eye, Orbit, and Eyelids, 1867, p. 202.

[2] Treacher Collins, Royal London Ophthalmic Hospital Reports, vol. xii. p. 345.

band of degenerating lens-fibres with spindle-shaped masses of material exhibiting transverse markings, while still further in the periphery lie sharply-outlined round cavities in the lens-fibres, filled with granular débris. Alt has also given us an interesting microscopic examination of such a case. The capsule, which was ruptured at the equator, was thickened and adherent to the conjunctiva, no normal capsular cells remaining. It was covered on the outside by a firm spindle-celled tissue with the remnants of blood-vessels. "There was granular clouding of the lens-fibres, with Morgagnian fluid and giant cells between the fibres." In a case of Riegel[1] there was firm vascular tissue binding the intact capsule to the conjunctiva, and a newly-formed epithelium lining the posterior inner surface of the capsule. Falchi[2] reports an instance where there was rupture of the capsule with marked proliferation of connective tissue within it. One of the most curious cases on record is that related by J. C. Wordsworth[3] where in consequence of a kick of a horse there was a dislocation of both lenses under the conjunctiva. A lens thus dislocated under the conjunctiva may for a long time remain visible as a more or less globular body, but usually diminishes in size by absorption, a process which takes place more readily when there has been a rupture of the capsule. Sometimes the dislocated lens undergoes degeneration and calcareous infiltration. (Arlt, Mitvalsky.) The lens is usually dislocated in its capsule, but it by no means follows that the capsule is everywhere intact, and Alt[4] and Sachs[5] have each demonstrated anatomically rents in such capsules. Mitvalsky,[6] who has reported thirteen cases of subconjunctival luxation, found that in one case only had the capsule remained behind in the eye. In some cases he found proliferation of the capsular cells and a gelatiniform degeneration of the cortical fibres.

### DISLOCATION INTO TENON'S CAPSULE.

In rare instances where the rupture of the globe takes place at or near the equator, the dislocated lens may lodge in Tenon's capsule (Wadsworth,[7] Montagnon,[8] Mueller,[9] and Schlodtmann).[10] Careful sections in Schlodtmann's case showed that the lens was dislocated in its capsule, that the subcapsular epithelium was in good condition, that the lens-tissue was undergoing granular degeneration and breaking down into albuminous clumps. There was a delicate web of newly-formed connective tissue between the pos-

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[1] Riegel, Archiv für Augen- und Ohrenheilkunde, 1877, Bd. vi. S. 8.

[2] Falchi, Nagel, Jahresbericht über die Leistungen und Fortschritte im Gebiete der Ophthalmologie, 1885, S. 367.

[3] Wordsworth, Royal London Ophthalmic Hospital Reports, 1882, vol. x. p. 204.

[4] Alt, Archives of Ophthalmology, 1877.

[5] Sachs, Archiv für Augenheilkunde, 1879, Bd. xx. S. 367.

[6] Mitvalsky, Archives d'Ophtalmologie, 1897, p. 348.

[7] Wadsworth, O. F., Transactions of the American Ophthal. Society, 1885, p. 144.

[8] Montagnon, Archives d'Ophtalmologie, 1877, p. 204.

[9] Mueller, Ruptur der Corneoscleral Kapsel, 1895, S. 18.

[10] Schlodtmann, Archiv für Ophthalmologie, xliv., 1, 127.

terior surface of the lens and the sclerotic, which was much more developed at either extremity of the equatorial region of the lens.

### DISLOCATION INTO THE VITREOUS.

Dislocation into the vitreous is the most common form of traumatic displacement, and in it, as in congenital and spontaneous luxation, the lens may remain transparent for some time in its new position, although it eventually loses weight, becomes smaller and more or less opaque. It may cause little disturbance in its new position beyond slight local atrophy of the chorioid and retina, or it may cause a serous chorioiditis, or, if pressing on the iris or ciliary processes, an attack of glaucoma. In time the vitreous breaks down more and more, and consequently the dislocated lens is apt to move about in the eye with various motions of the body and eyes, and at times when the patient bends forward to fall into the anterior chamber. Traumatic dislocation is usually monocular, but may at times affect both eyes, as in a case related by Lorch, where, after a fall on the back of the head, dislocation of both lenses followed. Where the lenses have been previously cataractous the immediate result of a traumatic luxation is often satisfactory. Janin,[1] for example, relates two cases where, in consequence of a fall, the cataractous lenses were dislocated into the vitreous, and in one of them both lenses were simultaneously displaced. Lawford[2] also gives the history of a case where, after three years of loss of sight from cataract, the opaque lens was luxated in a fit of coughing. The vision remained fair for twelve years, and then followed an attack of pain and tension on account of which the eye was enucleated. The lens was found to be shrunken and lying on the retina near the ora serrata, with a small patch of chorioidal atrophy adjacent to it. The angle of the anterior chamber was not obliterated, and there was an infiltration of small, deeply-staining cells around Schlemm's canal. As an instance of the curious lesions which at times accompany traumatic dislocation of the lens may be cited the case of Nettleship where, when the patient lay down, the opaque lens, which was usually in the anterior chamber, slipped up into a pouch between the sclerotic and the outer surface of the iris and ciliary body. The lens was extracted with slight loss of vitreous, but four months later the pouch still remained patulous. (Fig. 7.) Where the luxation is originally into the anterior chamber, or where, after a sojourn in the vitreous, the lens slips into the anterior chamber and becomes fastened in this position, we can observe a transparent body in contact with the cornea over a space of three or four millimetres, which gradually becomes

FIG. 7.


Dislocation of lens between the sclerotic and the outer surface of the iris and ciliary body. (Nettleship).

[1] Janin, Mémoires anat., physiol., et physiques sur l'œil et sur les maladies qui affectent cet organe, Lyon et Paris, 1772.

[2] Lawford, The Royal London Ophthalmic Hospital Reports, vol. xi. p. 327.

opaque, the cornea over it also becoming cloudy; and these symptoms are accompanied with considerable ciliary injection and watering of the eye. If not treated energetically, the cornea may either become staphylomatous or slough, and in the latter case the lens with the vitreous humor may be spontaneously evacuated. Graefe[1] relates an interesting case of dislocation into the vitreous, caused by the blow of a whip-lash, where the transparent lens finally settled into the anterior chamber so firmly that no shaking of the head would dislodge it. After a few months the lens clouded and the eye became irritable. After the use of cold compresses, Graefe tried a moderate discission. Fourteen days later there was a mass of exudation around the lens and opacity of the overlying cornea, although it was still smooth and covered with epithelium. On the following day the cornea sloughed, and the lens was spontaneously evacuated into the conjunctival sac through a three-millimetre opening in the cornea.

Prognosis in traumatic dislocation of the lens, whether complete or partial, is always serious. The injury is usually accompanied by so much hemorrhage into the interior of the globe and so much subsequent plastic inflammation and contraction as to cause eventually total loss of sight and shrinking of the eyeball. In rare instances, as in some of those above referred to, some useful sight is maintained, and in these cases vision is of course much improved by suitable convex glasses.

### THE TREATMENT OF LUXATION OF THE LENS.

The treatment of luxation of the lens will depend upon the position of the lens in the eye, the amount of injury or disease of the other structures of that organ, the degree of mobility which the lens possesses, and on its transparency or opacity. Usually in congenital luxation it is best to content ourselves with a correction of the refraction, being careful to do this with undilated pupil and with the head in the position in which the patient is likely to hold it during his work. In many cases where glasses do not afford the desired relief an iridodesis would seem to offer the typical measure of operative amelioration, securing a small and mobile pupil opposite to the centre of the lens, but the occasional occurrence of cyclitis after this operation usually deters us from employing it, and a small iridectomy, confined if possible to the sphincter of the iris, will at times be the best remedy, even if it does not afford as good a primary result as iridodesis. In these cases moderate and careful discissions, taking care if possible to avoid wounding the vitreous, will often cause a sufficient absorption of the dislocated lens to leave a clear pupil, and thus enable us to obtain good vision with a suitable glass. Where the lens is quite loose in the vitreous humor, even where there is fair vision, it is perhaps best to operate for its removal from the eye, inasmuch as the probabilities of the occurrence of glaucoma or serous chorioiditis or of breaking down of the vitreous and

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[1] Graefe, Archiv für Ophthalmologie, i., 1, S. 338, 344.

permanent lodgement in the anterior chamber become greater than those of undue loss of vitreous or of the awakening of inflammatory processes in a diseased eyeball by an operation. There will usually be loss of vitreous, but in most instances, by using a wire loop as traction instrument and gently pushing back the iris with it, the mass of vitreous which comes forward will carry the lens into the grasp of the instrument, provided it be perfectly free from all zonular attachments. If, however, there be any remnant of the zonule holding on to it, it will be still further displaced in the vitreous, and the portions of that jelly-like mass which come forward will press it backward into the eyeball. A realization of this state of affairs has made many operators try to bring the lens into the anterior or posterior chamber before attempting to extract it, and to prevent its escape into the vitreous during operation it has been proposed to operate on the patient in the prone position, he leaning forward over a table (Noyes), or to secure the lens in its position by inserting a bident behind it (C. R. Agnew), entering it into the ciliary body of one side and bringing it out in a corresponding position on the other side. The author is inclined to fear the bident, on account of the necessary wounds of the ciliary region, and believes that in the sitting posture, the patient leaning slightly forward, the manœuvre previously described will enable us usually to secure the evacuation of the lens. Unfortunately, owing to the increased dangers attending the administration of an anæsthetic in a sitting posture, and the deleterious effects of any struggling of the patient during its administration in displacing the lens, we usually have to forego anæsthesia, which, however, if pushed to absolute unconsciousness reduces the likelihood of loss of vitreous, except possibly in cases where there is severe vomiting during the recovery from its effects. Where the lens is firmly and permanently lodged in the anterior chamber there can be no doubt of the advisability of its extraction. This is best done by a small downward flap made just inside the limbus with a Graefe's knife. We must remember, however, that it may have become adherent to the cornea, and Mauthner reports a case where it had to be scraped away from the cornea with a spoon. The eye healed with a permanent and vascular opacity of the cornea.

In subconjunctival luxation, if the eye becomes quiet with any degree of vision it is usually best to allow the lens to remain in position. Where there are recurrent attacks of pain and cyclitis the eye should be enucleated. When we attempt to remove the lens it is well to wait until the sclerotic is firmly cicatrized, and to make the incision in the conjunctiva at the margin of the lens most distant from the rupture in the sclerotic, to avoid any chance of ciliary staphyloma following the operation. Lawson [1] relates a case of Bowman's where the removal of the subconjunctivally dislocated lens ten days after the injury was followed by the formation of a ciliary staphyloma at the position of the rent in the sclerotic; while Harlan [2] observed a

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[1] Lawson, l. c., p. 104.

[2] Harlan, Transactions of the American Ophthalmological Society, 1885, vol. iv, p. 140.

similar occurrence ensuing in removal of the lens five weeks after the injury. The same author observed a case where the lens remained for three years under the conjunctiva while the patient still had useful vision in the eye.

## ARRESTS OF DEVELOPMENT AND MALFORMATIONS OF THE LENS.

### ABSENCE OF THE LENS.

Morgagni, V. Walther, and Arnemann are all cited as giving examples of absence of the lens. This defect probably never occurs except either as the result of an arrest of development at an early stage of fœtal life, or as the consequence of disease. The cases recorded by Seiler and by v. Ammon as occurring in microphthalmic eyes appear to belong to the former category. Dunn[1] has published an interesting case of entire absence of the lens. There was a central cord in the vitreous corresponding to the position of the hyaloid artery. The cornea was unusually globose, but Dr. Dunn informs me that it presented no traces of scar or cicatrix of any kind.

### THE NORMAL LENS.

The normal adult lens is a biconvex body more curved at its posterior than its anterior surface, and measures on an average 9.5 mm. in its equatorial diameter, 4 mm. in its antero-posterior axis, with a radius of curvature of 8.2 mm. for its anterior and of 6 mm. for its posterior surface.[2] There is in healthy eyes some variation in measurement, but usually the size of the lens corresponds to that of the cornea and of the ciliary circle, being larger where these are large and smaller where they are small. The space between the equator of the lens and the ciliary processes in the vast majority of cases remains about the same, the diameter of the circle formed by the ciliary processes being usually about 11 mm. Slight defects in the distribution or in the patulousness of the fœtal blood-vessels, however, at times interfere with the development of the lens, and we find lenses which are small in comparison with the diameter of the ciliary circle of the eye from which they are taken.

### MICROPHAKIA.

Where abnormally small lenses exist there may be in consequence of similar arrests of development a relaxed suspensory ligament, or when, owing to disease of the eye, the connection between the zone of Zinn and the capsule is weakened or dissolved, the small lens, on any sudden arrest of motion of the eye or shock to the eyeball, readily prolapses into the anterior chamber, where it appears as a small, highly refractive crystalline disk, and is frequently by some similar process shaken back again into the vitreous chamber, or drawn there by gravity when, the pupil being

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[1] Dunn, Archives of Ophthalmology, January, 1896, p. 112.

[2] These dimensions are from Henle's measurements; the radius of curvature is from E. v. Jaeger.

fairly large, the patient assumes the recumbent position. Such lenses, however, as was shown in the previous section, may remain in their abnormal position for a long time, retaining their transparency for varying periods, but usually becoming cloudy and causing inflammatory opacity and softening of the cornea. Hartridge[1] gives an interesting account of such small but well-formed and transparent lenses existing in both eyes of a brother and sister. The eyes were highly myopic, and when the pupil was dilated by atropia and examined with the ophthalmoscope a considerable space was seen to intervene between the pupillary margin of the iris and the periphery of the lens. Mitvalsky also relates two such cases. In one of these the horizontal diameter of the cornea measured 12 mm., while after extraction the partially opaque lens measured only 7 mm. equatorially, by 4.5 mm. in the antero-posterior diameter. Such microphakic lenses are usually fairly well shaped, although perhaps more globular than usual.

### THE NOTCHED LENS—COLOBOMA LENTIS.

In such instances where the pupil is dilated with a mydriatic the ophthalmoscope shows a notch in the equator of the lens, usually so directed as to look downward, downward and inward, or downward and outward. The coloboma may vary in degree from a slight flattening of the convex edge of the lens to a deep concavity. Its edges are at times serrated and in rare instances we find two such indentations in the periphery. The equatorial notch probably always corresponds to some accompanying malformation in the zone of Zinn. At times it is accompanied with coloboma of the iris and chorioid, and it then corresponds in general direction with these defects. Notches are usually at the periphery of the lens, but the accompanying cut from Becker[2] shows that they may be confined to the posterior surface of the lens. (Fig. 8.) The figure represents a section of the lens of a new-born child suffering from congenital syphilis, with a curious horseshoe-shaped groove on its surface. The opening of the horseshoe was directed downward and the lens measured 7 mm. in equatorial diameter and 4.25 mm. in sagittal. In this case there was no defect in the zone of Zinn nor any other appreciable malformation of the eye.

FIG. 8

Horseshoe-shaped notch in the posterior surface of the lens. (Becker.)

A coloboma of the lens may be accompanied with an upward dislocation, with a corresponding defect in the zone of Zinn, as is well shown in the figure taken from a case reported by Gunn.[3] (Fig. 9.)

[1] G. Hartridge, Transactions of the Ophthalmological Society of the United Kingdom, vol. vi. p. 419.

[2] O. Becker, Atlas der pathologische Topographie des Auges, Tab. xii. Fig. 3.

[3] Gunn, Transactions of the Ophthal. Society of the United Kingdom, vol. ix. p. 166.

Fig. 10 depicts an interesting case of Christen.[1] In the right eye the iris was tremulous, and its tissue partly atrophic, although the pupil re-

Fig. 9.

Ectopia of both lenses, with coloboma of each lens and of the zone of Zinn. (Gunn.)

acted promptly to light. The lens was perfectly transparent, but partially dislocated inward and upward. The outer lower edge was flattened, and

Fig. 10.


Coloboma of right and left lens. (Christen.)

from it three threads of tissue extended outward towards the ciliary body. The left eye, also represented in the figure, in addition to a coloboma of the iris upward and outward, showed a flattened periphery of the lens at this point. The zonula could not be demonstrated in the coloboma. Dunn also reports an instance of notched lens with ectopia upward and inward. The diameters of the cornea were everywhere from one to two millimetres less than those of the well-formed fellow-eye. An interesting case is recorded by Baas[2] where the lens was kidney-shaped, the hilus being almost directly inward, and projecting into it was a plug of retained fœtal iris tissue which had apparently prevented a proper development of the lens in this direction. There may be a double notch in

Fig. 11.

Kidney-shaped lens, coloboma inward. (Baas.)

[1] Christen, Archives of Ophthalmology, June, 1896, p. 30.
[2] Baas, Klinische Monatsblätter für Augenheilkunde, 1893, S. 297.

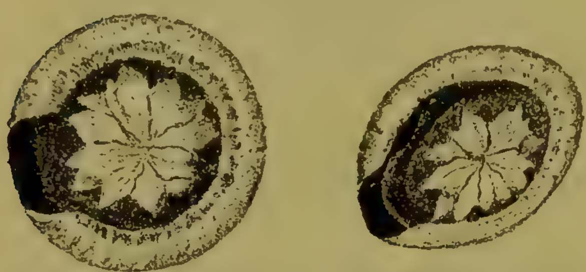
the lens, as in the cases recorded by Schiess-Gemuseus[1] and by Meyer.[2] In the latter case there were two distinct notches, one in the lower nasal and one in the lower temporal margin of the lens, of irregularly triangular form, whose apices reached about half-way between the equator and the centre of the lens. The intervening sector of lens tissue extended out to the normal position of the equator. The rest of the lens was normal in shape. The eye was myopic—a myopia probably due to the lens, — 2 D. giving V. = 1/6 when looking through the lens, while + 10 D. with dilated pupil, when the patient looked through the coloboma, gave equal acuity of vision. There was no coloboma of the iris or chorioid.

Coloboma of the lens is not necessarily connected with myopic refraction. Bowman and Heyl[3] each observed it in a hypermetropic and Gruening[4] in an emmetropic eye. Many cases of coloboma of the lens are now on record. Heyl (1875), in his paper, sums up twenty-three including his own, and in 1893 Bock[5] records forty-six. To these may be added (mostly since that date) those reported by Oliver, Iodko, Badal, Becker, Schiess-Gemuseus, Cissel, Stephenson, Marple, Hess, Theobald, and Rogman. Knapp gives a case where the defect was situated in the upper part of the lens, and Rogman has recently described another occupying the same situation. Iodko, Cartwright, Schaumberg, and Christen each report one where it occupied the upper outer part. Bowman, Baas, Schiess-Gemuseus, and Hess each record a defect pointing inward, and Schaumberg a second case where the notch was upward and inward, Lang and Theobald each describing a defect pointing outward. In Theobald's first case, where the notch was at the outer part of the lens, it was possibly traumatic, as was a corresponding scar in the cornea. In all other reported cases the defect has been in the lower part of the lens, or in its lower inner or lower outer portion. Of course most of these observations have been made with the ophthalmoscope, but notched lenses have also been extracted from the living eye and examined anatomically. Thus, Bowman[6] extracted a notched and dislocated lens on account of repeated attacks of glaucomatous tension, and describes it as "altogether too small and too convex. Its equator was circular but irregular, in a way to give indication of the tri-linear arrangement of its segments,—*i.e.*, it approached in a slight degree to a triangular outline when seen from the front or back." Badal[7] also extracted a notched lens in its capsule from an eye with coloboma of the iris and chorioid. Its horizontal diameter was eight millimetres, its vertical diameter at the notch seven millimetres, its sagittal five millimetres. As

[1] Schiess-Gemuseus, Archiv für Ophthalmologie, 1888, xxi. S. 453.
[2] Meyer, Bulletin de la Société Opht. de Paris, November, 1892.
[3] Heyl, Report of Fifth International Ophthalmological Congress, 1875, p. 16.
[4] Gruening, Report of Fifth International Ophthalmological Congress, p. 28.
[5] Bock, Die Angeborenen Colobomae des Augaepfels, Wien, 1892.
[6] Bowman, The Royal London Ophthalmic Hospital Reports, vol. v. Part 1, p. 16.
[7] Badal, Gazette des Hôpitaux, 1880, p. 459.

previously mentioned, the defect may be accompanied by a coloboma of the iris or chorioid, but is more often observed without any trace of such accompanying lesion. It usually affects both eyes, but in a number of cases the fellow-eye has been found entirely normal. In many instances it is recorded that the notched lenses were absolutely clear, but there have often been observed in them various degrees and forms of cataract. In Knapp's case the opacity was central, in that of Schiess a central capsular spot, in that of Cartwright a posterior opacity with retained hyaloid arteries; in one case of Bowman, in one of Gunn, in two of Rogman, and in one of Hess there was marked perinuclear (zonular) opacity. Fig. 12 shows the location of the notch and the form of the opacity in the left eye of the case of Hess. The fellow-eye, also represented in Fig. 12, presented zonular opacity without any coloboma of the lens. Bowman, in the paper already referred to, says, "the inner margin, instead of being a regular curve, presents several sinuosities, and takes, on the whole, a horizontal direction, as though the lowest part had sprung upward from its attachment to the ciliary body, while the lateral parts are still held by the suspensory ligament." While this feebleness or defect in the zonula is probably the true explanation of some superficial notches, and possibly due to imperfect closure of the fœtal slit, the deeper notches in the lens, as has been pointed out by Heyl and Hess, are probably caused by some congenital defect dependent upon an anomalous distribution of the hyaloid artery or of its branches to the posterior lens capsule. The exact nature, however, of such a congenital defect is very differently understood by the two authors just mentioned. Heyl holds that insufficient or arrested development of some of the vessels of the vascular lens-capsule causes an arrest in the development of the lens, while Hess maintains that the defects are due to pressure on the lens by some portion of vascular fœtal tissue which has not undergone regressive changes and absorption at the usual time. Bock also believes that the vascular plug of ingrowing mesoderm is a hinderance to development at the point of contact, while Bach[1] thinks that the lens at times becomes too large for the secondary optic vesicle, and that at the points of contact with the ingrowing mesoderm there is sufficient pressure to cause molecular degeneration of the subcapsular fibres, which undergo gradual absorption and leave a notch in their place.

Fig. 12.




Perinuclear opacity in each lens, with lateral coloboma of the left; coloboma of the iris in both. (Hess.)

[1] L. Bach, Archiv für Ophthalmologie, xlv., 1, S. 65.

## LENTICONUS.

The anomalies classed as lenticonus consist of a more or less conical projection of the anterior or posterior surface of the lens. Such formations give rise to various degrees of myopia in various areas of the pupillary space, the refraction being highest at the apex of the cone, thus producing a state of affairs which as regards the refraction of the eye much resembles conical cornea. In several of the reported cases the defect has been confined to one eye only. Webster[1] in 1875 gives a most interesting case where the apex of the lens cone projected into the anterior chamber. His picture of it is reproduced in Fig. 13. With normal pupil a glass —1½ gave V. = 20/CC, while with dilated pupil the periphery of the lens was so flat that + 1/10 gave V. = 20/XL. In 1880 Van der Laan[2] describes a protuberance in the anterior portion of the lens occupying about one-fourth of its surface. In the centre there was high myopia and in the periphery hypermetropia = 3 D. Lindner reports a case of lenticonus anterior with ectopia and corectopia. Both lenses were dislocated upward and had pyramidal projections on their anterior surfaces while the pupils were drawn downward.

FIG. 13.

Lenticonus anterior. (Webster.)

F. Meyer[3] in 1888 describes a case of posterior lenticonus in which there was a small opacity at the posterior pole. Knaggs,[4] Venneman,[5] Eiseck, Mitvalsky, Gullstrand, Salzmann, Doyne, Hartridge, L. Mueller, and Jackson[6] have also observed cases of lenticonus posterior. In describing his case Venneman remarks that in a year the central myopia became greater, this change corresponding with increased acuity of vision. He therefore hoped to see the lenticonus eventually change to a lentiglobus. Van der Laan also in the case above cited asserts that the lens cone was of gradual formation and had lasted eight years. It is therefore probable that in some cases the pathological changes initiated during the development of the eye do not become stationary but go on to further development. Weeks[7] and Roosa[8] each describe a case of posterior lenticonus in which there was opacity at the apex of the cone. In the patient of Weeks there were remnants of fœtal pupillary membrane in the other eye, which was

[1] Webster, Archives of Ophthalmology, iv. 2, 262.
[2] Van der Laan, Nagel, Jahresbericht, 1880, S. 364.
[3] F. Meyer, Centralblatt für praktische Augenheilkunde, February, 1888, S. 541.
[4] Knaggs, The Lancet, 1891, ii. p. 657.
[5] Venneman, Annales d'Oculistique, t. cv. p. 158.
[6] Jackson, Section on Ophthalmology, College of Physicians of Philada., 1894.
[7] Weeks, Archives of Ophthalmology, vol. xx. p. 260.
[8] Roosa, Treatise on Diseases of the Eye, 1894, p. 423.

otherwise healthy. In Roosa's case the fellow-eye was normal. Elschnig has also recorded a case of marked lentiglobus posterior with slight opacity of the parts involved.

In several of the cases of lenticonus there was a varying degree of opacity in the lens, but in the cases reported by Knapp, Venneman, Doyne, Hartridge, L. Mueller, Sym, and Cramer the lens appears to have been entirely transparent. In one of the two cases reported by Mueller, in spite of the reflex image from the anterior capsule being smaller in the centre of the pupil, the refraction in the axis of the lens was 2.5 D. less than in the periphery, while in his second case the usual relations obtained, the refraction being higher in the centre than in the periphery. Mueller believes the variation in the refraction in the different pupillary areas to be due to the shape of the nucleus of the lens, which according to him may be either spindle-shaped, giving higher central refraction, or saucer-shaped, giving (as in his first case) lower central refraction.

**Pathology of Lenticonus.**—With the exception of the case of Pergens,[1] who found lenticonus posterior in the buphthalmic eye of a four-and-a-half-year-old child, we have no anatomical investigations of this anomaly as occurring in man, and in default of this we must turn to similar conditions found in the lower animals. Becker[2] describes a conical projection on the posterior part of a rabbit's lens which was apparently everywhere covered with capsule and in which the lens-fibres although abnormally arranged were transparent. Hess[3] has since published accounts of two such malformations, one from the eye of a pig, the other from the eye of a rabbit. These are well shown in Figs. 14 and 15. In each there

Fig. 14.

Lenticonus in pig's eye. (Hess.)

Fig. 15.

Lenticonus in rabbit's eye. (Hess.)

is an irregularly globular projection from the posterior part of the lens covered by a very thin capsule, which in places is imperfect and wanting. The lens-fibres exhibited an abnormal course and marked granular degeneration, and in neither case was there any formation of a nucleus. The anterior portion of each lens was transparent, with normal capsule and capsular epithelium. In the case of the rabbit the fellow-eye presented a nuclear cataract. More recently Hess has had an opportunity of examining eight

[1] Pergens, Archiv für Augenheilkunde, xxxv., 1, S. 1.
[2] Becker, Anatomie der gesunden und kranken Linse, S. 125.
[3] Hess, Archiv für Ophthalmologie, xlii., 3, S. 234.

lenses where the nucleus was not central, but lay close to the capsule either at the posterior pole or between it and the equator. In some the outline of the lens was regular; in others the cone was caused by the nucleus projecting backward; in others again a layer of lens-fibres had applied themselves over the projecting nucleus. The posterior capsule was usually intact, but in a few instances was apparently ruptured in fœtal life between the posterior pole and the equator, and there was a rounded irregular mass of lens-fibres projecting backward out of the cleft. Bach[1] reports three cases of lenticonus posterior in the eyes of rabbits, in all of which the lenses were partly cataractous. In these he found a connective-tissue band, due to the thickened sheath of the central hyaloid artery, which by shrinking and pulling on the posterior capsule of the lens seems to have caused its thinning and rupture as well as either a conical or globular projection of the posterior part of the lens.

## DEFORMITIES OF THE LENS DUE TO PATHOLOGICAL PROCESSES WITHIN THE EYEBALL AND TO TRAUMATISM.

Every ophthalmic surgeon has certainly at times in examining enucleated eyeballs had an opportunity to see deformities of the lens, where, either from its elasticity or perhaps from pressure upon its equator, the lens, partially or completely dislocated, has become more or less globular in form, or where, owing to distention of the ciliary ring and pull on the zonula, it has become abnormally flat. Iwanoff[2] called attention to the softening and deformity of the lens in cases of suppurative panophthalmitis, and gives an illustration of it which is reproduced in Fig. 16, showing great swelling of its posterior surface. Hocquard[3] has recently written an interesting paper on this subject, and divides such deformities into two classes: first from pressure, second from traction on the zonula. He does not admit that even a young lens will materially alter its shape by simple relaxation of the zonula without pressure. Fig. 17 gives an instance of unusual flattening of the lens caused by distention of the eyeball in a case of staphyloma of the cornea with secondary glaucoma and marked increase in the diameter of the ciliary circle, while Fig. 18 represents the deformity of the lens caused by pressure from an intra-

FIG. 16.

Softening and deformity of lens in suppurative panophthalmitis. (Iwanoff.)

[1] Bach, Archiv für Ophthalmologie, xlv., 1, S. 59–60.
[2] Iwanoff, Archiv für Ophthalmologie, 1869, xv., 2, S 21.
[3] Hocquard, Archives d'Ophtalmologie, April, 1894.

ocular tumor. Ridley[1] found a circular projection on the anterior surface of the lens in two cases of glaucoma, which he thinks due to pressure of the sphincter of the iris on a softened lens. He terms this deformity "moulding of the lens;" while Fig. 19, from Becker,[2] shows a much distorted lens, the equatorial region of one side being much thinned and elongated, projecting into a staphyloma at the corneo-scleral junction. Ulcers of the cornea occasionally give rise to hernia of the lens. Where they are peripheral they are covered by iris, but when central the lens alone presents, and its transparent capsule and anterior layers, thus protruding, resemble very much a hernia of the membrane of Descemet.

FIG. 17.

Flattening of the lens in case of staphyloma of the cornea. (Hocquard.)

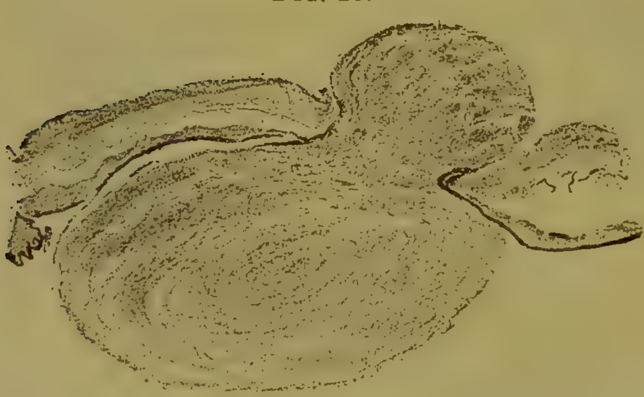
FIG. 18.

Deformity of lens caused by pressure of an intra-ocular tumor. (Hocquard.)

FIG. 19.

Distorted lens. (Becker.)

FIG. 20.



Hernia of the lens with intact capsule. (E. Treacher Collins.)

Fig. 20, from Treacher Collins,[3] gives an interesting example of such a hernia of the lens with an intact capsule. Of course any rupture or cut of the capsule, owing to swelling of the lens substance, gives rise to

[1] Ridley, Transactions of the Ophthal. Society of the U. K., xiv. pp. 138, 146.

[2] Becker, Anatomie der gesunden und kranken Linse, S. 125.

[3] Treacher Collins, The Royal London Ophthalmic Hospital Reports, vol. xii. p. 342.

irregular projections from its surface corresponding to the extent and location of the injury.

## CATARACT.

### A FEW WORDS ON THE VIEWS FORMERLY HELD IN REGARD TO OPACITIES OCCUPYING THE PUPILLARY SPACE, AND ON THE MEANING OF THE WORD "CATARACT."

In all ages opacities in and back of the pupillary space have attracted attention on account of the altered appearance of the eye and of the diminution of sight accompanying them. The color, consistence, and surface aspect of those pathological conditions in which the pupil had lost its normal black hue were designated with a variety of names in various languages, most of which were not only an attempt to describe those changes, but in many instances also an endeavor to indicate the opinion which was held as to their causes. The history of medical opinion, therefore, as to pupillary opacities, as given in detail by Sprengel, Hirsch, and Magnus, are interesting not only as showing the slow development of scientific knowledge, but also as affording most instructive instances of the dangers of theoretical reasoning on facts and data where we have no understanding of the minute anatomy and physiology of the tissues concerned. Magnus shows us that among the Egyptians, the Greeks, the Jews, and the Arabians all such opacities were classed as effusions, and in consequence hypopyon and inflammatory plastic exudations were confounded with the various opacities of the lens, and in some of the old books we are directed to shake the head of the patient violently to enable us to see whether the cloud is dissipated or altered in its appearance by this procedure. The Egyptian talked of "the rising of water in the eye," an expression which we find again in the Greek υποχυμα, from ὑποχεω (to flow from beneath); while the Hebrews spoke of "stationary water in the eye." The same idea is again encountered in the Latin word *suffusio*. Based on such ideas of pathology, their surgical remedial measures were of two varieties—preventive and curative.

**I. Preventive Measures.**—Among most of the nations of antiquity these procedures consisted of such a division of the vessels of the forehead and temple as would in their opinion prevent either a descent or a welling up of water within the pupil. This division was usually effected by incisions in the skin, carried down to the bone; and the subsequent healing and re-formation of the vessels was retarded by the use of tents or by scraping of the bone or by actual cautery. The Chinese and Japanese are said to have had a preventive surgical treatment consisting of acupuncture followed by actual cautery, believing that cataract and most other diseases are due to an interference with the circulation, which they try to stimulate in this manner.

**II. Curative Measures.**—These consisted in acupuncture to let the fluid matter out from the eye, or, if it proved stiffened and hard, to push it

out of the pupillary space, and where this proved impossible, to lacerate or incise the remnants in the anterior chamber or in the pupillary space. So have arisen the various methods of couching and depressing cataract, for which we find inexact descriptions in the earlier authors, but which in the time of Celsus had become definite and precise. The Indian method of couching as described by Breton[1] in the present day appears to have come down by example and practice from gray antiquity. The most astonishing circumstance in connection with these long ages of the practice of depression or dislocation of cataract is the persistence with which its practitioners held on to the theory of effusion, in spite of the fact that in many instances their daily practice must have convinced them when the lens broke up, or still more when it was dislocated into the anterior chamber, that it was really a solid, rounded lenticular body, and not a shrunken and condensed mass of pupillary effusion. Such pathological ideas, however, in more or less modified shape remained dominant until Brisseau, in 1705, read a paper before the Royal Academy of Medicine in Paris, in which he demonstrated to an unwilling and unconvinced audience that the true seat of cataract lay in the lens itself. A few years later, Maitre Jean,[2] who had already in 1692 convinced himself that the opacity lay in the lens, by observation of cataracts which he had luxated into the anterior chamber during attempts at depression, and especially by an autopsy of two eyes in which he had successfully performed the depression of cataract, published his work on Diseases of the Eyes, and succeeded better in arousing the attention of the medical world and convincing some at least of his contemporaries. The word "cataract" according to Hirsch[3] was first used in 1150 by Matteus Platearius of Salernum. According to some authors this is only another method of expressing a fall of water into the eye, they deriving the word from *καταρακτης*, a waterfall, and *καταρηγνυμι*, to break violently; while others insist that it comes from *καταρασσω*, to disorder violently.

## SYMPTOMS AND DIAGNOSIS OF CATARACT.

The word "cataract" has now come to mean any opacity in the lens or its capsule, although the latter variety is usually qualified by the word "capsular." The writers of the period when dislocation of the lens into the vitreous was almost the only operation practised for the relief of cataract were in the habit of calling the opacities which remained in the pupillary space capsular cataract; and Malgaigne in 1841 raised quite a storm of criticism by declaring that there was no capsular cataract. Although, as will be shown later and was first demonstrated by Arlt (1855), opacities and thickenings of the capsule can be observed in many cataracts, never-

[1] Breton, Trans. of Med. and Phys. Soc. of Calcutta, 1826, vol. ii.

[2] Antoine Maitre Jean, Traité des maladies de l'œil et des remèdes propres pour leur guérison, Troyes, 1707.

[3] Hirsch, Klinische Monatsblätter für Augenheilkunde, 1869, S. 284.

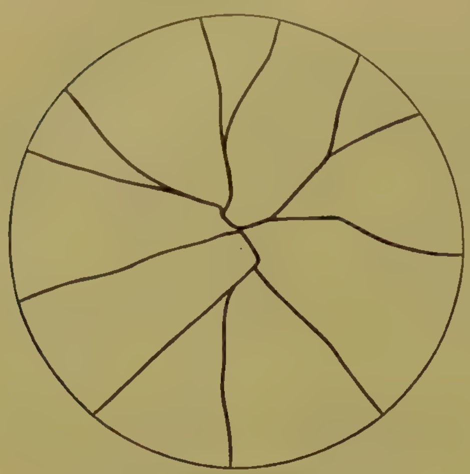
theless Malgaigne was right in believing that most of what was then thought to be capsular cataract was really part of the lens itself which had adhered to the capsule and had become opaque from the action of the aqueous.

### CONGENITAL AND TRAUMATIC CATARACT.

In congenital and in traumatic cataract we find partial and irregular opacities in various parts of the lens, but in the cataracts which develop in eyes previously normal and endowed with good vision the opacity usually commences either as opaque stripes in the equatorial cortical substance or as a perinuclear cloud. We will best understand the development of cataract if we glance at the lens at birth and study its growth and changes in adult life. According to Jaeger the average lens at birth measures 4.525 millimetres in antero-posterior, but only 6.345 millimetres in its equatorial diameter. In adult life the antero-posterior diameter remaining nearly the same, the equatorial has grown to 9.150 millimetres. In infancy the lens tissue is colorless and transparent and the index of refraction in the deeper parts of the lens is so nearly that of the superficial layers that it takes careful examination with oblique light to demonstrate its presence. During adolescence new fibres are constantly produced by the epithelium of the anterior capsule, which push their way around the equator and up towards the posterior pole, while the mass which constituted the entire lens at birth, having exhausted its developmental powers and pressed on by the newly formed fibres, becomes denser in consistence and assumes a pale straw-yellow color which is usually quite perceptible by the age of twenty. Owing to the presence of this dense centre the lens becomes less elastic and less capable of assuming greater curvatures of surface under the influence of the ciliary muscle. We find therefore, even in early childhood, as soon as children are sufficiently educated to be able to give reliable answers, that we can demonstrate a rapid diminution in the power of accommodation with a recession of the near-point. The growth of the nucleus continues steadily during health, in consequence of the deeper layers of cortical fibres, each in turn, becoming denser, more yellow, and adhering to the central mass. In fact, when in consequence of age or impaired nutrition this process stops, the first step has been taken towards the formation of a cataract, although in some cases, usually in advanced old age, after the entire lens has become sclerosed, we find it by transmitted light of a rich reddish-brown color with sufficient degeneration of the fibres to dim the images formed by it, and we have a state which, owing to its interference with distinct vision and to the small amount of light reflected from the pupil, is known as black cataract (cataracta nigra), a form, however, entirely different from that which is sometimes found after hemorrhages into the eye where blood-coloring matter has been absorbed by the lens. In adolescent and adult lenses there is considerable difference in the index of refraction of the different layers, and therefore, when we examine such healthy eyes with oblique

light and at the same time study the lens with a magnifying glass, we see the sectors appearing grayish and the intersectorial divisions as dark lines dividing them. The direction of these lines varies in different individuals and even in the same individual at different times of life, their ends becoming more branched as age advances. Fig. 21, taken from Tweedy,[1] gives a good example of the anterior stella of a healthy adult human lens as seen with dilated pupil and the use of a magnifying glass and oblique light. Fridenberg,[2] who examined many healthy lenses with binocular magnifying glasses and oblique light, found that out of one hundred lenses about one-fifth had four primary rays, two-fifths five rays, one-fifth six rays, and one-fifth seven rays.

Fig. 21.



Stella of normal lens. (Tweedy.)

*Entoscopic examination* casts a most interesting light on the structure of the healthy adult lens and displays its sectors as well as various shining or opaque dots throughout its substance, and almost every one who wears spectacles has frequent opportunity for studying his own lenses by the diffusion circles arising from some brilliant point of reflection on the frames of his glasses and consequently from a point inside of the range of distinct vision.

### THE DEFECTS IN THE TRANSPARENCY OF THE LENS WHICH MAY BE CONSISTENT WITH NORMAL VISION.

It is important to consider attentively these defects, first, as seen entoptically, and, second, as demonstrable by transmitted and reflected light. The lens spectrum is readily seen when we admit to the eye a small, intense beam of homocentric light such as is obtained by looking through a half-millimetre aperture in a metal plate on which a bright beam of light is concentrated by a condensing lens. Listing, Helmholtz, and Donders have given us instructive pictures of their own lenses as seen in this way, and describe, first, pearl dots with bright centre and dark edges or rims which lie mostly near the anterior capsule; second, opaque or slightly translucent spots which are sometimes round, at others irregularly angular; third, radiating lines which correspond with the intersectorial lines of the lens as seen with dilated pupil and oblique light when the lens is studied with a magnifying glass. Owing to the intense illumination obtained in the manner above described, the pupil contracts and the field is narrowed unless we have recourse to a mydriatic to hold the pupil dilated. This inconvenience may be avoided by the method which is hereafter described,

[1] Tweedy, R. L. O. H. Rep., vol. viii. p. 25.

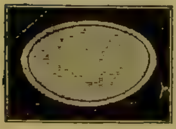
[2] Fridenberg, Archiv für Augenheilkunde, xxxi. S. 293.

because the comparatively feeble illumination and the dark room secure a semi-dilated pupil and wide field. The lens spectrum can then be more readily observed by taking as the sources of light the images reflected from the posterior and anterior surfaces of a convex lens of 20 D. If in a dark room with only a single source of illumination, and this placed behind and to one side of the observer, we regard these brilliant but minute images through a lens of — 20 D. held in a spectacle-frame close to the eye, we thus obtain a pencil of divergent rays, and the lens spectrum appears as a disk composed of bright dots lying close to each other but not in contact, the interspaces being comparatively less illuminated, and we usually find a few "pearl spots,"—that is, larger bright spots surrounded by a distinct dark ring. We also see bright radiating lines corresponding with the intersectorial lines of the lens, each line being slightly tortuous and in places more or less beaded in outline. If now we remove the concave lens from the frame and insert in its place a convex one of + 20 D., we obtain a pencil of convergent rays and a very different picture. What previously appeared as bright dots now look like clear, darkish round spaces surrounded by a bright rim. Both the smaller dots and the large pearl dots have this appearance. The radiating lines have almost disappeared, but attentive study shows that they may be traced as darkish stripes with brighter edges. In some lenses, however, the dark radiating stripes are bordered by sharp-cut bright lines on each side. All these appearances are best seen when the lens used in reflecting the images is held at such an angle as to appear as a faint whitish illuminated cloud, and we have a most instructive comparison of the brilliant image furnished by the reflection of the posterior curvature of the lens with a fainter image from the anterior surface. By slight movements of the lens which serves as the source of light, towards the observer's eye and away from it, we may make the dots and stripes alternately sharper and more visible. All these appearances are more instructive when we compare them with those of young and healthy lenses when examined with the ophthalmoscope, the pupil having been dilated and a lens of + 20 D. placed behind the mirror. Thus seen, every crystalline lens looks more or less granular, and in some are seen a few clear round spaces surrounded by a hair-like black outline. These are sometimes close to the intersectorial lines and send hair-like prolongations to them, but more frequently have their seat in some other part of the lens, usually close to the anterior capsule, and probably correspond with the pearl dots as seen entoptically. In examining numbers of presumably healthy young lenses it is astonishing how often slight but distinct remnants of the fœtal pupillary membrane are visible. In a series of eleven hundred and eighty-four consecutive cases who came for error of refraction in my hospital services and who were carefully examined for lenticular opacities with the ophthalmoscope armed with a 20 D. lens behind a mirror, seven had corneæ so cloudy that the state of the lenses could not be definitely ascertained. Of the remaining eleven hundred and seventy-seven, nine hundred and

sixty-nine had clear lenses, one hundred and sixty presented either minute ring spots or black dots, one had central anterior polar cataract, eighteen showed striæ of commencing cortical cataract, while in twenty-nine there were present traces of persistent pupillary membrane. As regards the time of life of individuals examined, nine hundred and thirty-three were under thirty years of age, and of these one hundred and thirty presented either rings or black spots in the lens, while the striæ of incipient cataract were found only in those over thirty years of age, and eighteen times in two hundred and forty-four individuals. According to Pulvermacher,[1] in sixty children under ten years of age with entirely normal eyes the magnifying-glass behind the mirror showed in twenty-six—*i.e.*, forty-three per cent.—lens opacities, consisting of distinct spots and vesicles.

Besides the above-mentioned clear dots and granular appearances we often find irregularly-rounded minute and partially opaque spots, which presumably also are indications of want of absolute transparency in the lens tissue, probably congenital and not due to post-natal pathological change. When the lens is studied by a strong magnifying-glass and oblique light, we obtain a grayish reflex from its substance, while the intersectorial lines appear dark, and some of the large ring-shaped bodies previously described appear grayish or whitish. Fig. 22, from Donders[2] (reduced one-half size), gives a good view of the lens spectrum from the right eye of this illustrious observer at the age of forty-five. Darier[3] has lately published instructive entoptic pictures of his own lenses and those of some of his friends. He employs to produce them a concave lens of 30 to 40 D., through which the experimenter looks at a candle distant five metres. After adult life the lens grows but slowly. The accompanying diagram, Fig. 23, from Priestley Smith,[4] shows the average increase of size between the ages of twenty-five and sixty-five in lenses which have remained healthy and transparent. The increase in size with advancing years is accompanied by a steady gain of weight. Fig. 24, taken from the same observer, is so arranged as to show the size and weight of lenses at different ages by their displacement of water. The figures at the left-hand side of the diagram indicate the weight as shown by displacement of water, while the ages are indicated in the line on the top of the figure. One hundred and fifty-six lenses taken from the eyes of ninety-one individuals were examined, and it appears that the weight of the lens increases 4.5 milligrammes in each decade of life. The dots in the figure indicate clear lenses; the circles, lenses which were found to be more or less cataractous. The same author[5] shows that lenses which have become cataractous are smaller and weigh less than

Fig. 23.



Growth of lens between the ages of twenty-five and sixty-five. (Priestley Smith.)

[1] Pulvermacher, Centralblatt für prakt. Augenheilkunde, 1894 (Supplement).
[2] Donders, Anomalies of Accommodation and Refraction, p. 200.
[3] Darier, Ann. d'Oculistique, September, 1895, p. 198.
[4] Priestley Smith, Trans. Ophth. Soc. U. K., vol. iii. p. 88.
[5] Ibidem, p. 85.

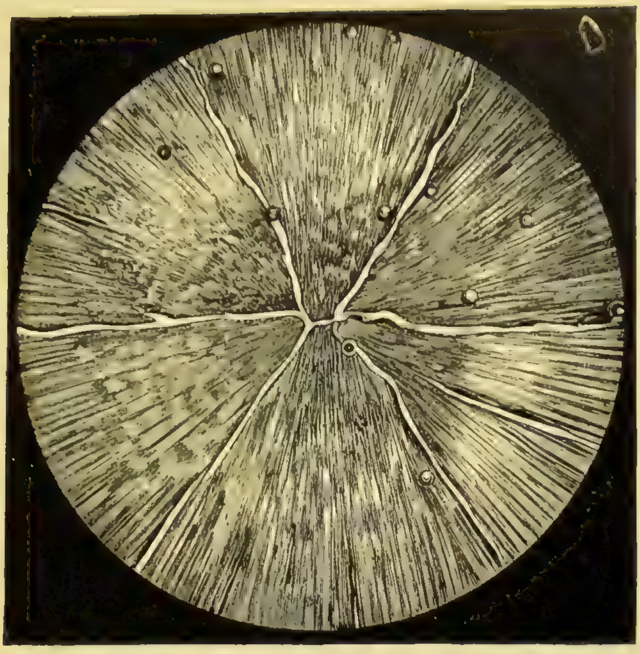


FIG. 22.—Lens spectrum. (Donders.)

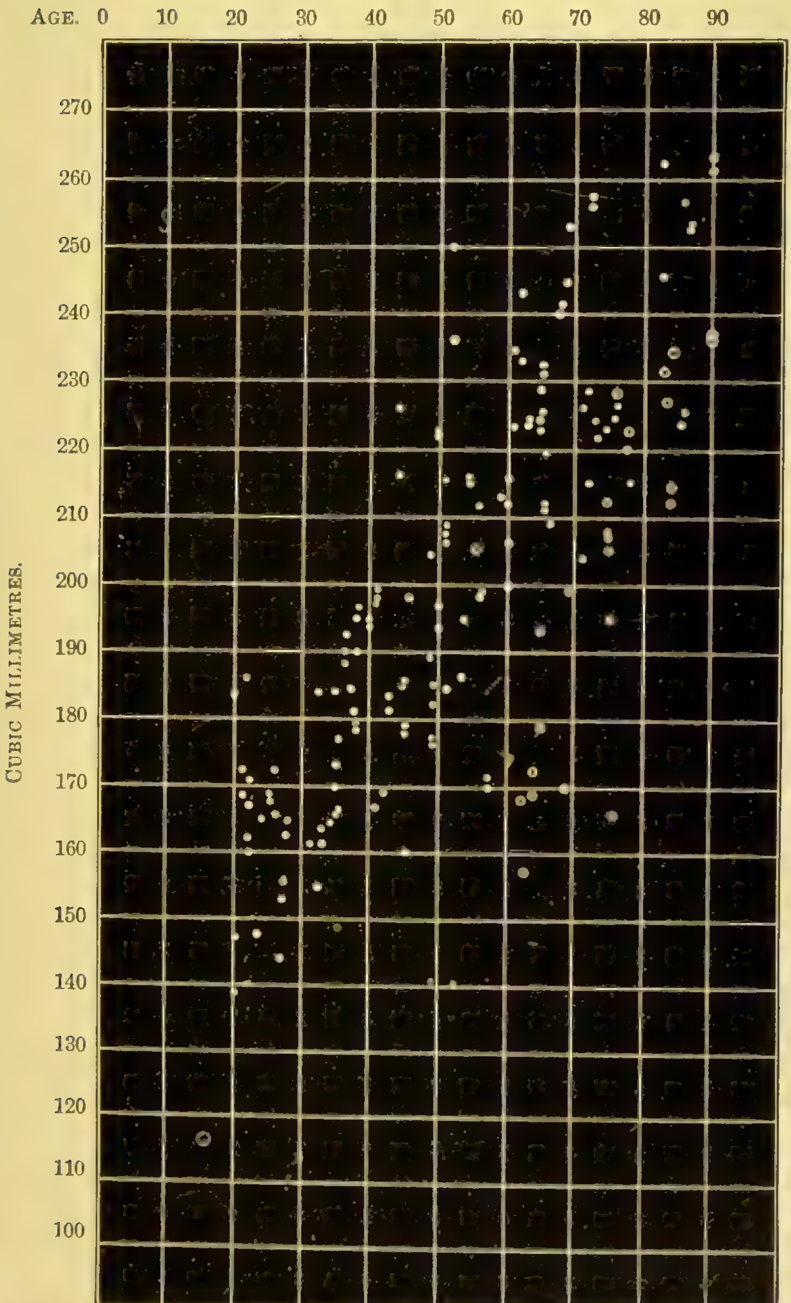



FIG. 24.—VOLUME OF LENS.—One hundred and fifty-six lenses from the eyes of ninety-one persons Weight of lens increases about four and five tenths milligrammes in each decade. Dots show clear lenses; circles show cataractous lenses. (Priestley Smith.)

do healthy lenses at the same time of life. These statements have been confirmed by Collins,[1] who tells us that the average weight of ten clear lenses was 0.204 gramme, while the average of six with beginning cataract was 0.113 gramme. When, owing to failure of the general health or pathological changes in the vessels of the interior of the eyeball and the gradual exhaustion of nutritive force in the capsular cells, the lens ceases to increase in size and density, then the newly-formed fibres are no longer pushed tightly up against the nucleus, and spaces arise between the lens-fibres which become filled with serum. They are usually first formed in the cortical substance, either just outside of the equator of the nucleus or in the extreme periphery of the lens, in the position where growth is normally most active. The fibres of the cortical masses themselves swell slightly from imbibition of this endosmosed fluid and commence to undergo molecular degeneration, and when these processes manifest themselves in the part of the lens corresponding to the pupillary area, they deflect and impede the passage of light to the retina, and we have a diminution in the acuity of vision. At this stage patients turn to the doctor for relief, and examination shows them to have incipient cataract. The subjective complaints which lead patients to seek relief are usually those of early fatigue of the eye, dim vision, motes or specks before the eyes, and the seeing small and distant but brightly illuminated objects irregular in outline and double. The patient complains perhaps that his glasses no longer suit him, and that the moon looks unnaturally horned or double. Examination with the test-letters will show that in strong light the acuity of vision is still not far from normal, and that perhaps the correction of some small astigmatism produced by the unequal swelling of the lens will improve it. The defects of sight are more tangible in near work, and while glasses enable the patient to read fluently ordinary print, still Jaeger 1 is not deciphered at the proper distance and print of any size soon tires the eye. When, owing to swelling of the anterior cortical, the iris is pushed forward and the anterior chamber reduced in size, the strong light of the sun out-of-doors becomes very disagreeable to the patient, and he usually avoids or moderates it by the use of a shade and dark glasses. Day by day and month by month, as the lens becomes hazier, the dimness augments and amount of useful vision decreases until, as the cataract ripens, a dense fog settles down on all objects and even bright sunlight has a moonlight pallor. Even after all useful form vision has disappeared, patients will often recognize large patches of bright and well-marked colors, although in many the great yellowness of the nucleus diminishes their power of distinguishing the lighter shades of blue. Even in advanced cases the patient is still able to perceive bright points of light and to recognize the shadows of objects held in front of him. Besides the slight irritability of the eye above mentioned and faint sensations of uneasiness and discomfort, there is usually no pain in uncomplicated cataract.

[1] Treacher Collins, Ophthalmic Review, 1889, p. 321.

When we examine objectively eyes affected with incipient cataract there is usually a marked diminution of the normal blackness of the pupil, and often a small pupil with a narrow anterior chamber. The pupillary reactions are prompt, but the dilatation of the pupil under influence of a mydriatic is almost always less than that produced in healthy eyes of persons of the same age, and in many instances, owing apparently to some degree of atrophy of the iris tissue, a dilatation of four to five millimetres is all that can be obtained, and this dilatation is often accompanied in such cases by marked pericorneal flushing of the eyeball and of the bulbar conjunctiva. When we look into the pupil thus distended we obtain a gray or grayish-yellow reflex most marked from the deeper layers of the lens, while if we inspect the pupillary area minutely with a magnifying-glass we often obtain a mother-of-pearl glitter from the fibres of the anterior cortical, while the striation of the lens-substance is so arranged that the individual sectors show distinctly. Gray spots and streaks are often visible, which may be rounded, ovoid, linear, or irregularly pyramidal, the latter being usually situate in the periphery of the lens. They lie in either the anterior or the posterior cortical or in both, sometimes next the capsule, sometimes deeper, usually at the extreme periphery of the lens, or at times near the equator of the nucleus. The exact shape of all such opacities is still better seen by using + 16 D. or + 20 D. lenses behind the ophthalmoscopic mirror, and we can thus discern that the spots and streaks are irregularly rounded, spindle-shaped, or pyriform, while we can usually see throughout the lens infinitesimally small dotted opacities, more marked in the cortical layers. The opacities which appeared gray by oblique light now show as dark spots on a yellow-red field, as they intercept and deflect from our eyes the returned light from the illuminated eye-ground. As the cataract increases in density less and less light penetrates the fundus and is returned from it, so that eventually all red reflex from the eye-ground is lost. The accompanying cut,[1] Fig. 25, shows an exceptional case where the sectors had become opaque, but red light still came through the intersectorial lines and through the periphery of the pupil. As the cataract ripens the lines become opaque and show with reflected light as dense white lines, while the anterior cortical becomes more and more opaque, until finally the posterior pupillary margin of the iris appears to rest absolutely on the opacity of the anterior cortical and the clear space separating the latter from the uvea has disappeared. At this stage the lens has generally lost its swelling and the anterior chamber has reacquired its normal size or perhaps become a little deeper, and the cataract is said to have become ripe. All cataracts, however, do not reach this stage, but some remain always limited to the perinu-

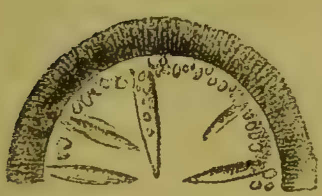
FIG. 25.

Sectors of lens cloudy with transparent intersectorial lines. (Tweedy.)

[1] Tweedy, R. L. O. H. Rep., vol. viii. pp. 24–36.

clear layers, so that, examined with the ophthalmoscope, they show a grayish-black mass lying deep within the lens, with a cortical that is so clear that with dilated pupil the red light from the eye-ground returns freely all around the periphery of the lens. The above-described changes are so important and have been so well studied and pictured by Jaeger and by Magnus that graphic illustrations of the latter author are here reproduced. Fig. 26 (Taf. III. of Magnus) shows a case of commencing cataract where irregularly round, ovoid, or pear-shaped figures may be seen in the equator of the lens, with large pointed shoots advancing towards the pole. Fig. 27 (Magnus, Taf. IV.) shows a still more advanced form of equatorial cortical cataract where the individual spaces of degeneration are larger, and where there are also round and ovoid figures in the cortical nearer the pole, while some of the spaces between the sectors show as strings or lines of varying diameter. It has been the experience of the author that cortical cataract in the majority of instances commences in the inner lower quadrant

FIG. 26



Incipient cataract. (Magnus.)

FIG. 27.

Incipient cataract. (Magnus.)

FIG. 28.

Incipient cataract. (Magnus.)

of the lens. Fig. 28 (Taf. XI. of Magnus) represents slight opacities in the equatorial cortical, and much more marked linear, spindle, and irregular ones in the zone surrounding the equator of the nucleus, while irregular dots and elongated figures occupy the more central cortical. Cataract may also commence in the anterior or posterior pole of the lens, usually either as irregularly shaped masses of opacity or as linear or oval figures. Where such masses are at the posterior pole they very commonly have star-like prolongations in the posterior cortical, extending outward towards the equator of the lens. When this is the case the eye-ground should be carefully examined for retino-chorioidal changes, if this is still practicable with dilated pupil, and if not, the field of vision carefully tested. According to Magnus the first appearances of senile cataract are to be found in the cortical substance close to the equator in 82.69 per cent. of all cases; in the poles of the lens in 9.61 per cent., and in the perinuclear cortical in 7.69 per cent., and daily clinical experience bears him out in the statement that the first indications of change are most often found in the peripheral posterior cortical. Where long, broad shoots intrude on the pupillary space oblique light and a magnifying-glass will at times show a folding of the lens-capsule. A ripe cataract usually presents slight clouding of the thin layer of pupillary anterior

cortical, while the large, yellow, waxy-looking nucleus comes up close to the surface, and at this time or a little later white opaque spots are often formed immediately under the capsule and in the superficial layer of lens-fibres. These are caused by epithelial proliferations with subsequent degeneration of the cells and are known as capsular cataract. After this stage is passed the fibres of the nucleus undergo a little further granular degeneration, while the cortical softens and either remains slightly yellowish or becomes in some instances entirely opaque, whitish, and milky. Where this softening proceeds to any considerable extent we have a loose nucleus floating about in the capsular sac, hidden usually more or less completely by the white and fluid cortical. By tilting the head forward we may often cause the nucleus to approach the anterior capsule sufficiently to permit its yellowish color and some part of its outlines to be distinguished. This stage is called *Morgagnian cataract*, and an admirable colored illustration of it is given by Beer.[1] Sometimes cholesterine crystals form in the degenerating lens-matter, and we have the curious spectacle of a shining, glittering mass of crystals within the lens-capsule. Where in the very commencement a cataract becomes whitish and milky, we have good ground to suspect not only that the exterior layers are fluid or semi-fluid, but also that there is accompanying disease of the interior of the eye. When the degenerative changes in the lens which have produced cataract go on and the patient lives long enough, in some instances further changes may occur which result in restoration of sight. The lens may so far liquefy and absorb as to leave only a small remnant of it in the capsule, or the capsule may burst and the semi-liquid lens-matter thus discharged into the aqueous may absorb, or, as we have already seen in speaking of dislocation of the lens, there may be a spontaneous sinking of it into the vitreous humor. The two former methods of termination will be more minutely discussed under the head of "Second Sight." It must, however, by no means be supposed that the above-described changes in the lens follow each other without halt or in any given period of time. Nothing can be more uncertain and various than the rate of development and formation of cataract. It is often for long periods nearly stationary, and then once more, at times without any evident cause, goes on to further development. Of course, any severe sickness or any intercurrent inflammation of the eye tends to hasten its development. For practical purposes, however, we must not rest satisfied with the diagnosis of cataract and of the stage of its development, but must endeavor to find out the state of the optic nerve, retina, and other structures in the interior of the eye, in order to ascertain whether its removal is likely to be of advantage to the patient. In the very earliest stages, where the opacities are mainly situated in the periphery of the lens, we may study the eye-ground with the ophthalmoscope, and any one who conscientiously looks for such lenticular changes in senile eyes will be surprised to see how very fre-

[1] Beer, Lehre von den Augenkrankheiten, Bd. ii., Wien, 1817.

quently they occur, even in eyes which have given their possessor no inconvenience, and in which there are no other pathological changes visible with the ophthalmoscope. In more advanced cataract the view of the fundus oculi will either be insufficient or entirely shut off, and we must rely on the external appearances of the eyeball, its tension, and the promptness of light perception and extent of the field of vision for further information as to the condition of the interior of the organ. The cornea should have its normal polish and transparency. In many instances it may have marked arcus senilis without any detriment to the course of healing after operation. Any disease of the conjunctiva or of the lachrymal sac is a most unfavorable complication. The iris should not only be prompt in its reaction to light, but the pupil should dilate readily and to a fair extent under the action of a mydriatic. A sufficient degree of atrophy of the muscular constituents of the iris to prevent fair dilatation is an unfavorable sign, indicating often other changes and degenerations in the uveal tract. Fulness of the anterior scleral veins and a tension either much below or much above normal are also unfavorable signs. The most important tests of the condition of the interior of the eye are the promptness of light perception and the extent of the field of vision. In most ripe cataracts, in a perfectly dark room, a glimmer of light can be perceived from a candle-flame held at a distance of fifteen to twenty feet. In some very dense opacities, such as the early stage of Morgagnian cataract, we may have to approach the light to a distance of ten or twelve feet before it is recognized, and the eye may, nevertheless, have a fairly healthy retina and optic nerve. The examination of the cataractous eye by the use of Foerster's apparatus to determine the amount of light perception would probably give more accurate results; and, according to Trompetter,[1] when the eye is placed in such an apparatus at a distance of eighteen inches from a normal candle, about half such eyes perceive the light of an eighteen-millimetre square opening at first, but after sojourn in the dark the perceptive power rises, and light from an eight-millimetre square opening is recognized. Where the opening has to be increased to thirty-two millimetres square to obtain light perception, the cataract is certainly complicate. Under any circumstances, however, a fairly intelligent patient will, in spite of the great dimness and blurring of the image, appreciate that two sources of light exist when two candles or wax tapers are held at a distance of eighteen inches in front of his eye and then are separated from each other. Where he is made to fix one of these, a second shine or glimmer will usually be perceived from the other as long as the moving candle still throws a sharp and distinct image on his cornea. When any difficulty as to obtaining good fixation exists the patient can be helped by making him point with his finger towards the central candle, or, if necessary, he can be made to hold it himself. In this way we not only measure the field of vision, but by asking which is the brighter light can obtain some idea of the state of cen-

[1] Trompetter, Klinische Monatsblätter für Augenheilkunde, 1880, S. 84.

tral vision. If the peripheral light be the more distinct, we have either an unusual density of the nucleus with comparatively clear peripheral cortical,—a state of affairs which we ought readily to recognize by the use of oblique light,—or else we have some diminution of central vision due to changes in the interior of the eye. Of course, such information, as far as we can obtain it, is most valuable, and certainly every surgeon of experience has at some time been disagreeably surprised by finding that after a successful operation for the removal of cataract no useful vision exists, owing to extensive macular changes. The diffusion of light by the opaque lens is so great that we cannot even with the greatest care map out minute changes in the interior of the eye or even demonstrate the blind-spot; but we can at times recognize coarser changes, such as a considerable detachment of the retina or the total abolition of central vision.

### ON THE CATARACT PRODUCED BY THE INGESTION OF POISONOUS DOSES OF SUBSTANCES INTO THE SYSTEM, OR BY THE DIRECT INTRODUCTION OF NOXIOUS MATERIALS INTO THE ANTERIOR CHAMBER.

It has long been known to physiologists that toxic doses of various substances introduced into the general system either subcutaneously, or by direct injection into the blood, or by ingestion in the stomach, will at times produce cataract. Thus, Kunde,[1] in 1857, shows that either common salt or nitrate of soda introduced into the large intestine or under the skin of frogs would produce cataract, and that the same substances caused similar effects in kittens and in dogs. He also found that strong solutions of sugar under like circumstances produced cataract, and that in frogs the opacity of the lens would clear up when the animal was again put into water. Anatomical examination showed him that fluid-filled spaces had formed between the lens-fibres, and that the fluid in them had a different index of refraction from that of the lens-fibres themselves. Kuehnhorn,[2] in 1858, substantiated these experiments, and attributed the cataract to the influence of these substances in extracting water from the lens. Mitchell[3] agreed as to the facts, but argued that it cannot be the abstraction of water which causes the opacity, inasmuch as he had found that the exposed lens when dried does not become opaque. The anatomical basis for the early stages of the clouding of the lens thus artificially produced appears to be an œdema, raising the capsule in places and separating the lens-fibres, this being accompanied in a later stage by proliferation of the epithelial cells and by granular degeneration of the lens-fibres. All experimenters have found that a similar superficial clouding of the lens takes place when it is removed from the eye and placed in a solution of sugar, and that an immersion in water once more clears it. Deutschmann[4] maintains that under

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1 Kunde, Zeitschrift für wissenschaftliche Zöologie, Bd. viii. S. 466.

2 Kuehnhorn, De Cat. Aquæ inopia effecta, Gryphiæ, 1858.

3 S. Weir Mitchell, Amer. Journ. Med. Sciences, 1860, pp 106–110.

4 Deutschmann, Archiv für Ophthalmologie, 1877, xxiii., 3, S. 112.

these circumstances the lenses only cloud when the solution contains at least two and one-half per cent. of salt or five per cent. of sugar. Heubel[1] claims that all materials which have a strong affinity for water when introduced into the circulation produce cataract, and that Guttman is mistaken in supposing that potassium chloride and calcium chloride do not. The former passes off rapidly by the kidneys, and both soon prove fatal to the animal by heart-paralysis; but both, when introduced into the conjunctival sac or into the anterior chamber, cause prompt clouding of the lens. Heubel, by such local application, produced thirty cataracts in animals, and, extracting these lenses, compared them with the sound lenses on the other side. He found that in all cases the cataractous lens contained less water than its fellow on the other side, and that the more extensive the opacity the greater was the loss of water. He maintains that similar processes take place in all animals, differing slightly in form according to the normal build of their lenses and the direction of the sectors. Similar opacities to those produced by the articles mentioned result from the use of menthol and naphthalin. Bouchard,[2] in 1887, was the first to call attention to the interesting effects on the eyes caused by the ingestion of NAPHTHALIN. Kolinsky,[3] in 1889, gives us a minute study of its effects. He found that it acted on all inward parts, the smaller the animal the greater the effect of any given quantity of the drug. The cataract appears always to be secondary, and dependent upon changes in the ciliary body, the vitreous, the retina, and the chorioid. The first lesions observed are hemorrhages into the ciliary body and into the chorioid. These are accompanied by œdema of the retina and lens, and later by separation of the vitreous and of the retina. When large doses are administered the lens swells quickly, and fluid is found between the capsule and the lens-fibres, with degeneration of the cortical fibres. Naphthalin cataract, when once started, may go on to become more dense without any further administration of the drug. Once fairly developed, it never entirely clears up, but always leaves some opacity behind. In the later stages crystals of phosphate of lime are deposited in the lens. There is œdema of the retina and lens, with marked proliferation of capsular epithelium and the formation of giant cells. Magnus[4] found that when he gave rabbits three to four grammes of naphthalin for each kilogram of weight, the first appearances of cataract could be sometimes seen after six hours. These appearances consisted of transparent stripes in the lens, corresponding to deeper grooves in the lens substance. A little later peripheral cloudiness sets in in the anterior and posterior cortical. There was marked proliferation of epithelium, and there were fissures between the fibres filled with cloudy fluid. According to Magnus, the lenticular opacity thus produced may disappear

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[1] Heubel, Archiv für die gesammte Physiologie, 1879, xxi., 5, S. 253.
[2] Bouchard, Recueil d'Ophtalmologie, 1887, p. 91.
[3] Kolinsky, Archiv für Ophthalmologie, xxxv., 2, S. 29.
[4] Magnus, ibidem, xxxvi., 4, S. 150.

in eighteen days. The same author found that the use of ten to twenty grammes of common salt produced clouding behind the equator in four hours, while in a dog or in a cat one hundred grammes of grape sugar produced cataract, the opacities showing themselves at the equator. Hess agrees in the main with the foregoing statement, and describes the first signs of naphthalin cataract as fine, clear shoots in the equatorial parts of the lens, which soon become cloudy and coalesce with neighboring stripes, so that in ten days the entire subcapsular cortical may become evenly cloudy. Magnus, Kolinsky, and Hess all agree that in the early stage of naphthalin cataract there is great congestion of the ciliary body and processes, and it is possible that by the altered secretions of these parts the pabulum of the lens becomes abnormal and its nutrition impaired. Klingmann,[1] also, insists that there are always symptoms of iridocyclitis before the slightest clouding of the lens takes place. It should be also stated that naphthalin usually produces a violent catarrhal diarrhœa in the animal to which it is administered.

**Cataract Caused by the Ingestion of Ergot.**—The frequency with which cataracts are met in some local areas immediately after an epidemic of poisoning by ergot would seem to indicate that this drug has a similar power of disturbing the nutrition of the lens. Meyer[2] describes an epidemic in Siebenbürgen where the symptoms were violent cramps of the muscles, with consecutive contraction, anæsthesia of the feet, and, as a later symptom, cataract. The cataracts formed slowly, always in both eyes, and a large proportion occurred in young people. He attributes this to spasm of the intra-ocular muscles. Longetsnikoff reports seventy-one cases, and also attributes the formation of the opacity of the lens to contraction of the intra-ocular muscles. Telpjaschin[3] saw in one locality, one year after an epidemic of ergot poisoning, twenty-seven cases of cataract, most of them in individuals under thirty years of age. Kortneff, after an epidemic in the district of Nolinsk, found that cataracts thus produced required from three months to one year to become ripe, and that those occurring in individuals over thirty years of age had the appearance and consistence of senile cataract. The eye-grounds were pallid and the vessels contracted during the period of convulsions, but at other times there was often hyperæmia.

## THE PATHOLOGY AND PATHOLOGICAL ANATOMY OF CATARACT.

A short study of some of the facts as to the nutrition of the healthy lens will enable us to understand more readily the phenomena of disease. In post-natal existence the lens, deprived of the blood-vessels which have

[1] Klingmann, Ueber die Pathogenese des Naphthalinstars, Archiv für path. Anat., cxlix. 1.

[2] M. Ignaz Meyer, Archiv für Ophthalmologie, viii., 2, S. 120.

[3] Telpjaschin, Medizinisknop Oborrense, xxxi., 5, S. 525. Quoted by Schoen.

nourished it in the fœtal state, depends for its maintenance and growth entirely on the absorption of fluid from the surrounding parts. It is evident that the lens, owing to its anatomical build, with prismoid and band-like fibres lying in close juxtaposition, would act like so many thin glass plates in sucking up a capillary layer of fluid from any surrounding fluid medium. These being all enclosed by an animal membrane,—the capsule of the lens,—we would have acting the ordinary laws of osmosis, and the amount of fluid taken up or given out would vary with the density of the fluids within and without the membrane, and the amount and kind of matter appropriated would, as in other tissue, depend on the vital activity of the cells composing it. The experiments of Deutschmann [1] have shed much light on this process and the channels in which nutritive materials in the lens are most readily taken up and transmitted through its structure. He administered fifteen grammes of potassium iodide to a rabbit, killed it after three hours, and found that when the lens, enclosed in its capsule, was laid in a solution of platinum chloride the ensuing reaction showed that the subcapsular capillary layer of fluid beneath the posterior capsule and the adjoining layers of the posterior cortical, as well as the entire equator of the lens, were the parts most strongly impregnated with the iodide. The subcapsular layer of the anterior capsule showed the same reaction in a less degree, while the anterior cortical and the nucleus did not exhibit any traces of it. He further found that when salt was placed under the skin of the back in frogs the resulting cataract commenced in the equator. Other investigators have since attained somewhat different results, but all agree that the points of most ready absorption are in the equatorial region of the lens. Thus, Ulrich,[2] using the ferrocyanide of potash and tincture of the chloride of iron as his staining material, could only find proofs of its absorption at the equator of the lens. Schöler and Uhthoff,[3] using fluorescin, insist that the fluid enters only by the route of the zone of Zinn and the canal of Petit through the equator, and that fluid never passes directly from the vitreous to the lens According to Magnus,[4] vesuvin injected into the carotids appears in the eye, finding its way along the zonula and entering the lens at the equator. Samelsohn,[5] after study of the distribution of fine particles of iron rust in lenses in which splinters of iron had for some time been embedded, concludes that the nutrient stream enters the lens at the equator, curves through it centripetally, collecting again near the anterior pole to be eliminated in the region where the anterior fibres of the zonula are inserted into the capsule. The exact method of egress of fluid from the lens is less well made out. Schöler and Uhthoff, in their paper on the effect of fluorescin, absolutely deny that any

[1] Deutschmann, Archiv für Ophthalmologie, xxv. 2, S. 227.
[2] Ulrich, ibidem, xxvi., 3, S. 33–82.
[3] Schöler and Uhthoff, Jahresbericht der Schölerischen Klinik, 1881.
[4] Magnus, Deutsche med. Wochenschrift, Nr. 40, 1881.
[5] Samelsohn, Klinische Monatsblätter für Augenheilkunde, 1881, S. 265.

coloring matter ever escapes through the posterior capsule into the vitreous. Morano claims to have demonstrated pores in the anterior capsule, but their occurrence has not been confirmed by other observers. Becker, who worked for years at the anatomy of the lens with indefatigable industry, states that he has looked for them in great numbers of thin sections, but has always failed to find them. It is possible that further investigation may show channels of escape along the insertion of the zonular fibres. H. Bence Jones[1] has made an admirable study of the rate of absorption of crystalloids in healthy and in cataractous lenses. He found that where three grains of lithium chloride were introduced into the stomach of a young pig, in half an hour lithium could be demonstrated in the lens, while in an old pig, after the lapse of a similar interval, none was to be found in the lens, thus demonstrating the more rapid absorption in the young. He further showed that absorption took place in old, and even in cataractous lenses, although more slowly, and that the drug was afterwards slowly eliminated. Thus, when twenty grains of lithium carbonate were administered to a cataract patient twenty-five minutes before operation, no trace of lithium could be demonstrated in the lens, while in other cases where the same quantity was ingested two and a half hours before operation lithium could be demonstrated in the watery extract of the lens. If three and a half, four, five, or seven hours were allowed to elapse between ingestion of the drug and extraction of the cataract, lithium could be readily demonstrated in every particle of the lens, while if a period of seven days was allowed to elapse, it had been entirely eliminated, and not a trace was demonstrable in the cataractous lens when extracted.

The equatorial region, where nutrition in the lens is most active, is also the place where disturbances in the nutritive processes first manifest themselves.

### MICROSCOPIC CHANGES IN CATARACTOUS LENSES.

Foerster[2] was the first to give an accurate description of these changes by examining with moderate magnifying power the lenses of seventy-two eyes with commencing cataract in individuals between fifty-four and eighty-seven years. The lenses were obtained after death and placed in hollowed glass slides and watch-glasses, immersed in vitreous humor, and closed in with a thin cover-glass. He found the opacities forming a thin layer over the comparatively clear and yellow nucleus and extending as a narrow zone around and on both sides of the equator of the nucleus. There were four principal forms of opacity: "first, short and narrow white lines, which formed a broken circle around the nucleus; second, thin white clouds, which either extend from one side of the equator to the other or appear in detached masses on each side; third, white stripes which run in a meridional direction close to the nucleus, and are broadest and thickest at the equator of the

[1] Jones, Proceedings Royal Society of London, vol. xiv. pp. 400–424; also Preliminary Report, 1865, p. 221.

[2] Foerster, Archiv für Ophthalmologie, iii., 2, S. 187, 198.

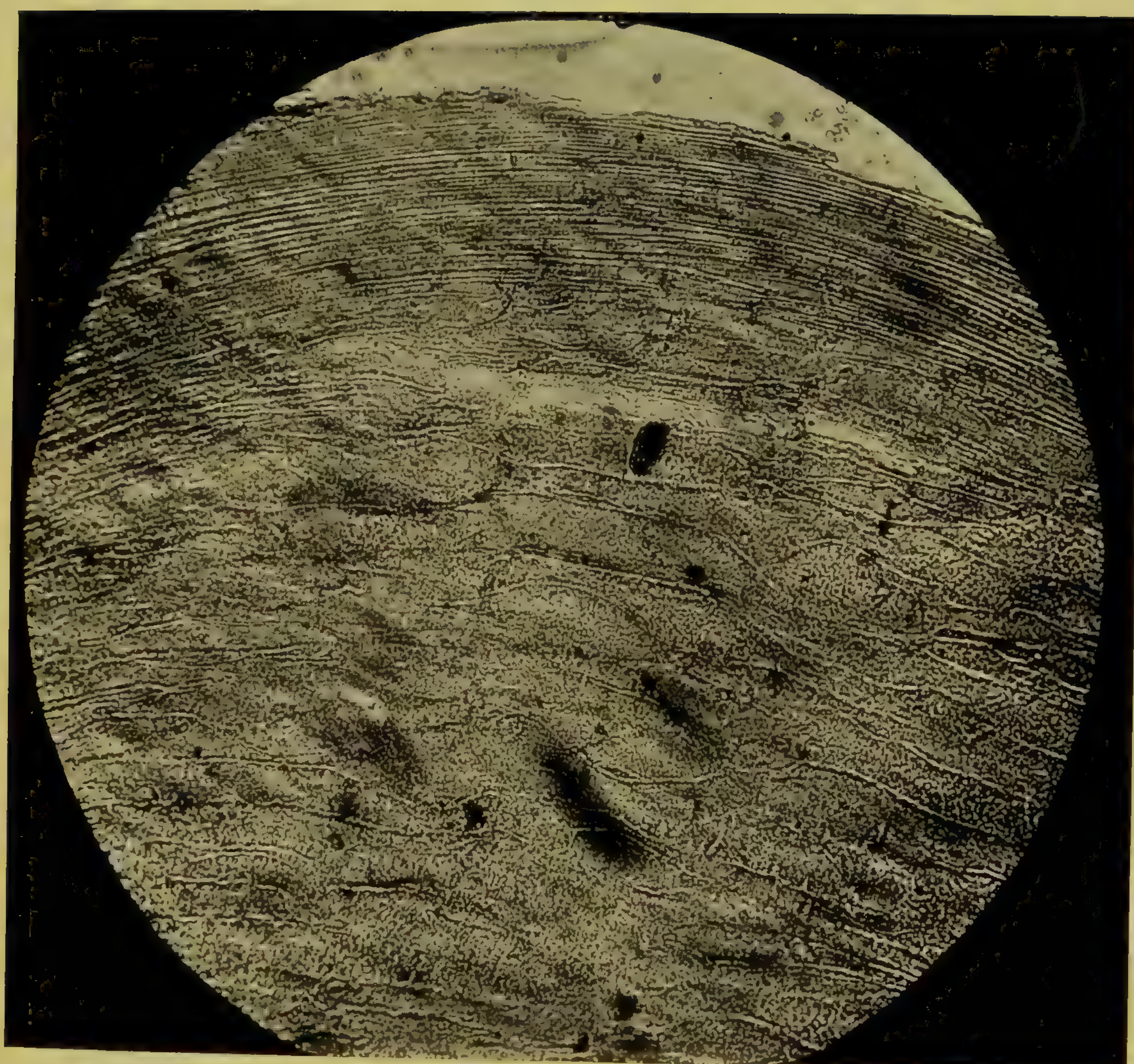
nucleus, becoming pointed towards the pole; fourth, cloudy, indeterminate forms which encircle the whole nucleus. The concentric clouds increase, and invade the entire thickness of the cortical as well as the superficial layers of the nucleus, but it is especially the striped opacities which increase in number and strength until they destroy the entire cortical." Out of the entire number of lenses thus examined there was but one with a purely perinuclear opacity, and it was taken from a myopic eye with a visual axis of twenty-nine millimetres. Foerster calls attention to the similarity in position of the perinuclear opacities of commencing senile cataract to that occupied by the stationary ones of zonular cataract. The stage of cataract thus accurately pictured by him is a considerably later one than that in which the ophthalmoscope shows us the first signs of opacity in the equator of the lens, and the fact that in some instances in the earliest stages we can alternately make the conical opacities there found appear, by varying the direction of the light, either dark or translucent, shows that we have to do with a phenomenon of total reflection, and that such appearance may therefore be due to spaces filled with fluid of a different index of refraction from the lens-fibres. Where the clouding of the lens is spreading throughout the cortical we have an increase in the size of the lens and of its watery contents, and the anterior chamber becomes smaller. This is clinically recognized as the stage of swelling, and the increase of size is supposed by Becker to be due exclusively to shrinking of the fibres and accumulation of fluid between them. I am inclined to believe with Knies, that in commencing cataract there is a molecular granulation with increase of size in each individual fibre, but my pathological material having been derived largely from lenses obtained by extraction, most of my sections exhibit a more advanced period of cataractous degeneration, and I have rarely had an opportunity of studying the process at its very incipiency. This stage of swelling is followed by a loss of fluid and a return of the lens to its normal size, known clinically as the stage of maturity of the cataract, while later the lens shrinks still further, and whitish dots and streaks form in and under the capsule, due, as we shall presently see, to the formation of capsular cataract. In cataractous lenses Foerster describes cracks between the concentric layers of fibres which are filled with a molecular substance. Becker has figured the same appearances, and insists that similar ones, less in size, may be met with in the healthy lenses of the young, the only distinction being that the molecular contents in youth stain vividly, while in cataractous lenses these masses are less stained than the surrounding lens-fibres. This distinction can scarcely be relied on as a criterion, as in many sections of well-marked cataract such molecular material stains much more intensely than the surrounding lens-fibres.

Every one, I imagine, who has examined many cataractous lenses has at times seen such appearances. The molecular degeneration of the lens-fibres is their first microscopic evidence of cataractous change, and occurs in very various degrees in different zones on the same lens. In senile cata-

ract (Fig. 29) the lens-fibres near the capsule are usually the last to present any considerable degree of change, while those lying between these and the nucleus exhibit far more advanced degeneration, while in cataract due to intraocular tumors and to effusion of plastic lymph on the anterior capsule the molecular degeneration is apt to commence close to the capsule (*vide* Fig. 59, p. 312). The formation of spindle-shaped spaces between the fibres is beautifully shown in Fig. 30, where the granular contents of the spaces have stained much more deeply than the surrounding material, have pushed the adjacent lens-fibres aside, and caused them to assume a wavy and undulating course. The cavities in this instance are most numerous in the perinuclear cortical. The fibres themselves are everywhere granular (*vide* Fig. 29), and the edge of the fibre seen in section often appears as if made up of minute beads or globules, while the granulation in the interior of the fibre is usually finer, and the tumescent and degenerating lens material causes the fibres to exhibit in section irregular swellings. In places these appear to have nuclei and resemble large vesicular cells (*vide* Figs. 29, 32, and 33). The internal structure of the fibre is often fissured by lines which run across it, causing it to appear as if it was broken up into irregular-sized segments. Fig. 31 shows this as seen by an immersion-lens, and Fig. 32 represents a similar state of affairs seen under low power.

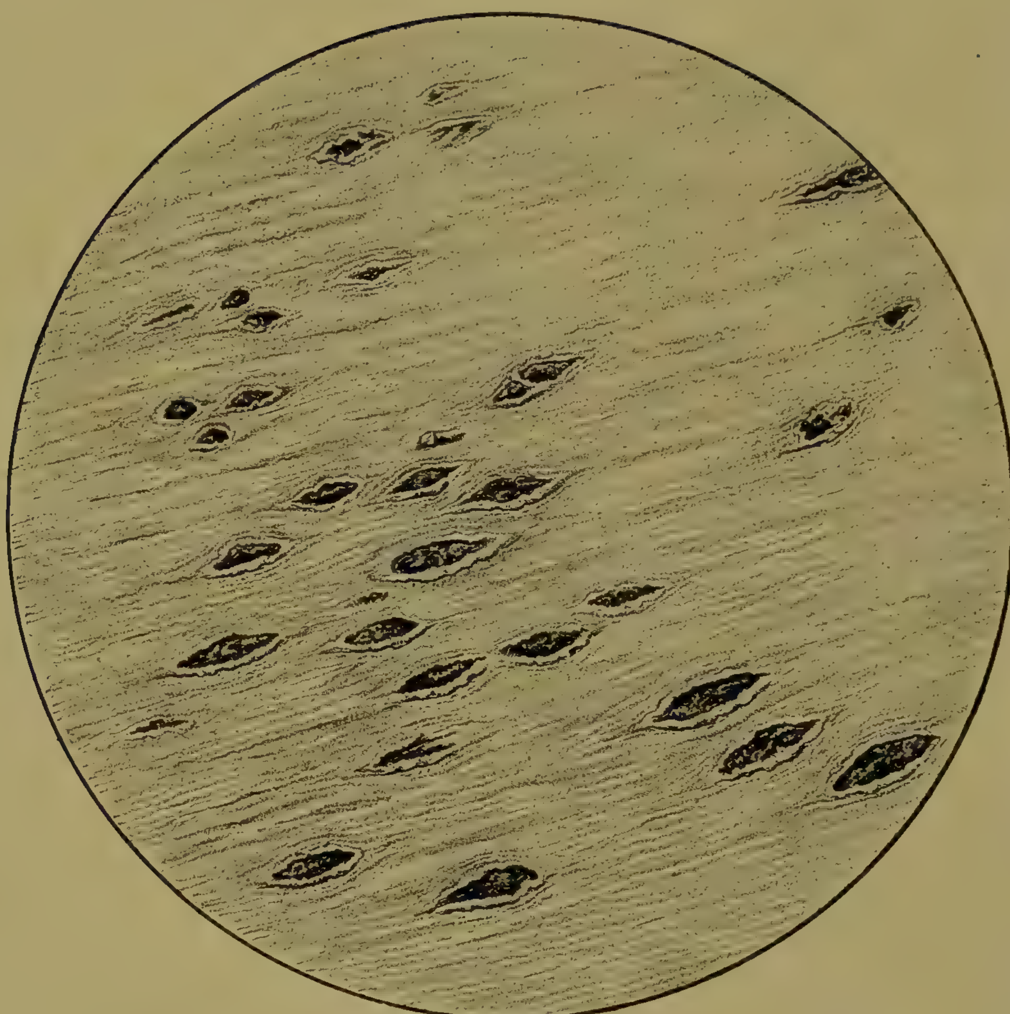
Much less advanced changes are shown in Fig. 33. This section exhibits moderate molecular changes in the lens near the vortex, and the fan-shaped extremities of the fibres have been pushed from their insertion in the capsule by proliferating epithelium. The deeper-lying layers are much more opaque and granular, and in some of the deeper-lying fibres nuclei may be seen. Foerster claims to have demonstrated slight changes in the nucleus of cataractous lenses. Becker maintains that the nucleus is not involved in such cases, but remains clear. My own specimens, which are thin sections made with microtome from lenses imbedded in celloidin, show slight molecular and granular degeneration with occasional dots of highly refracting material. Among the earlier changes in the concentric cortical lens-fibres may be mentioned a state of hyaline degeneration, which I have encountered only in lenses in which there has been posterior synechia, or in eyes with increased pressure, and in which degeneration is most marked immediately under the synechia. Fig. 34 represents this change in the external layers immediately under the capsule. The lens is partly cataractous, and was found in an eye which was in a state of glaucoma produced by a sarcomatous tumor of the chorioid. In this instance the hyaline changes which are confined to the external layers may possibly have been due to pressure. In the peripheral layers of the concentric lens fibres we often find large spindle-shaped swellings of the lens-fibres, each with a nucleus, sometimes elliptical, as in the healthy lens-fibre, and sometimes rounded. Such swellings, when large, look like vesicular cells. Besides these formations, and often near to them, we find large interfibrillar spaces filled with an albuminous material, which is divided by transverse fissures, and which

Fig. 29.



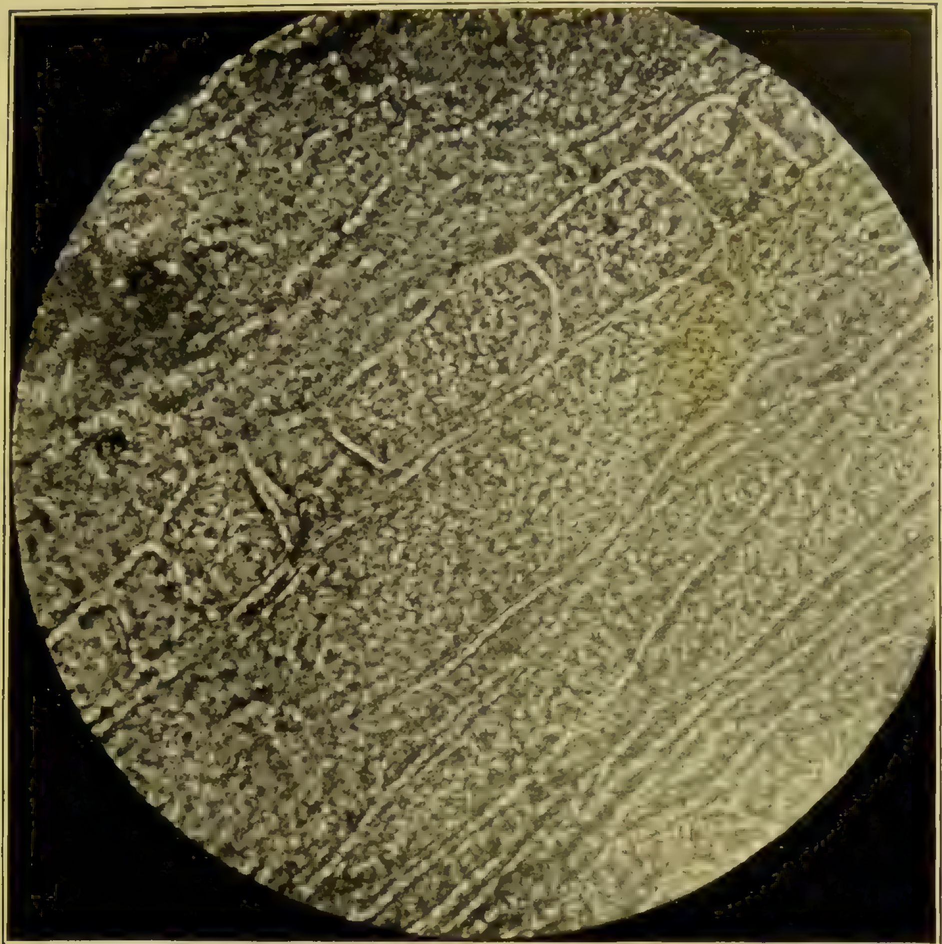
Section of senile cataract. (Photographed by Dr. George W. Norris.)

Fig. 30.



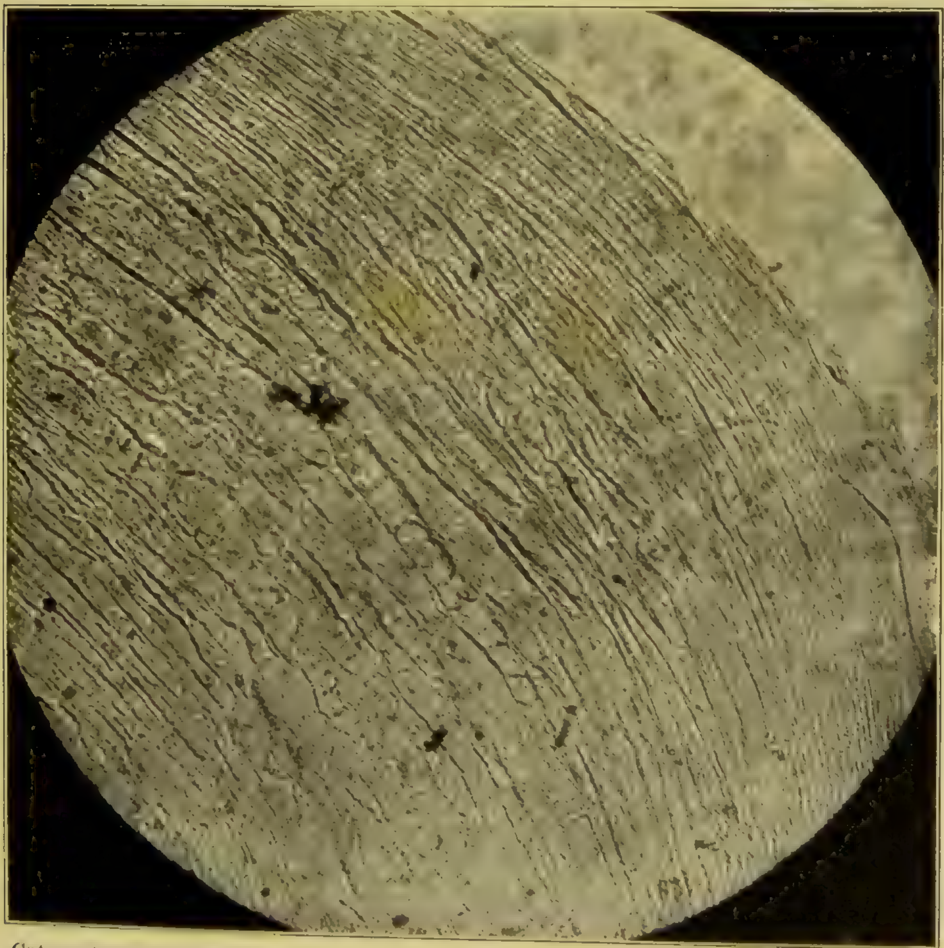
Spindle-shaped spaces between lens-fibres filled with molecular material.
(From a drawing after a photograph by Dr. James Wallace.)

Fig. 31.



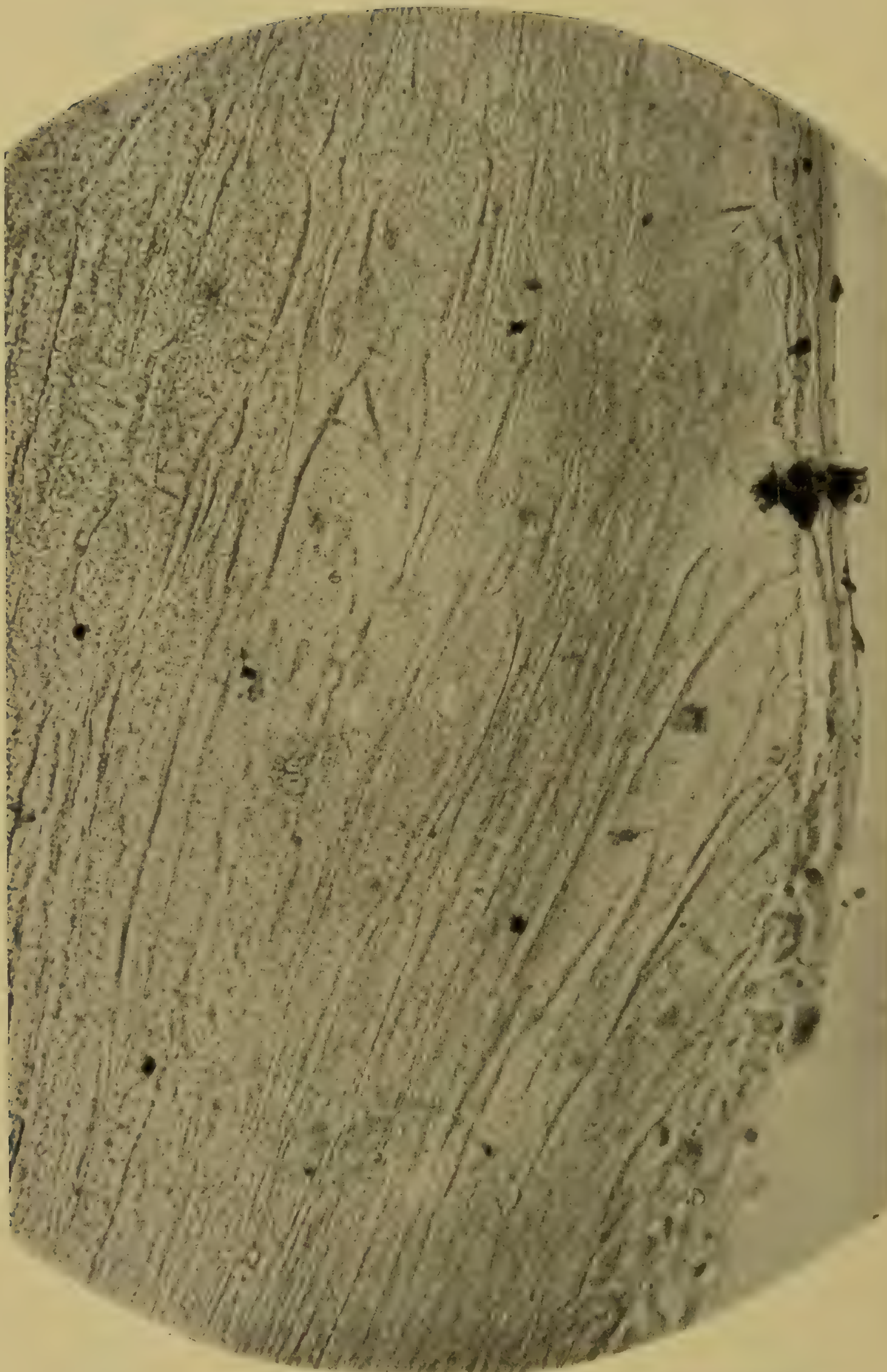
Cataractous degeneration of lens-fibres. (Photographed by Dr. George W. Norris.)

Fig 32.



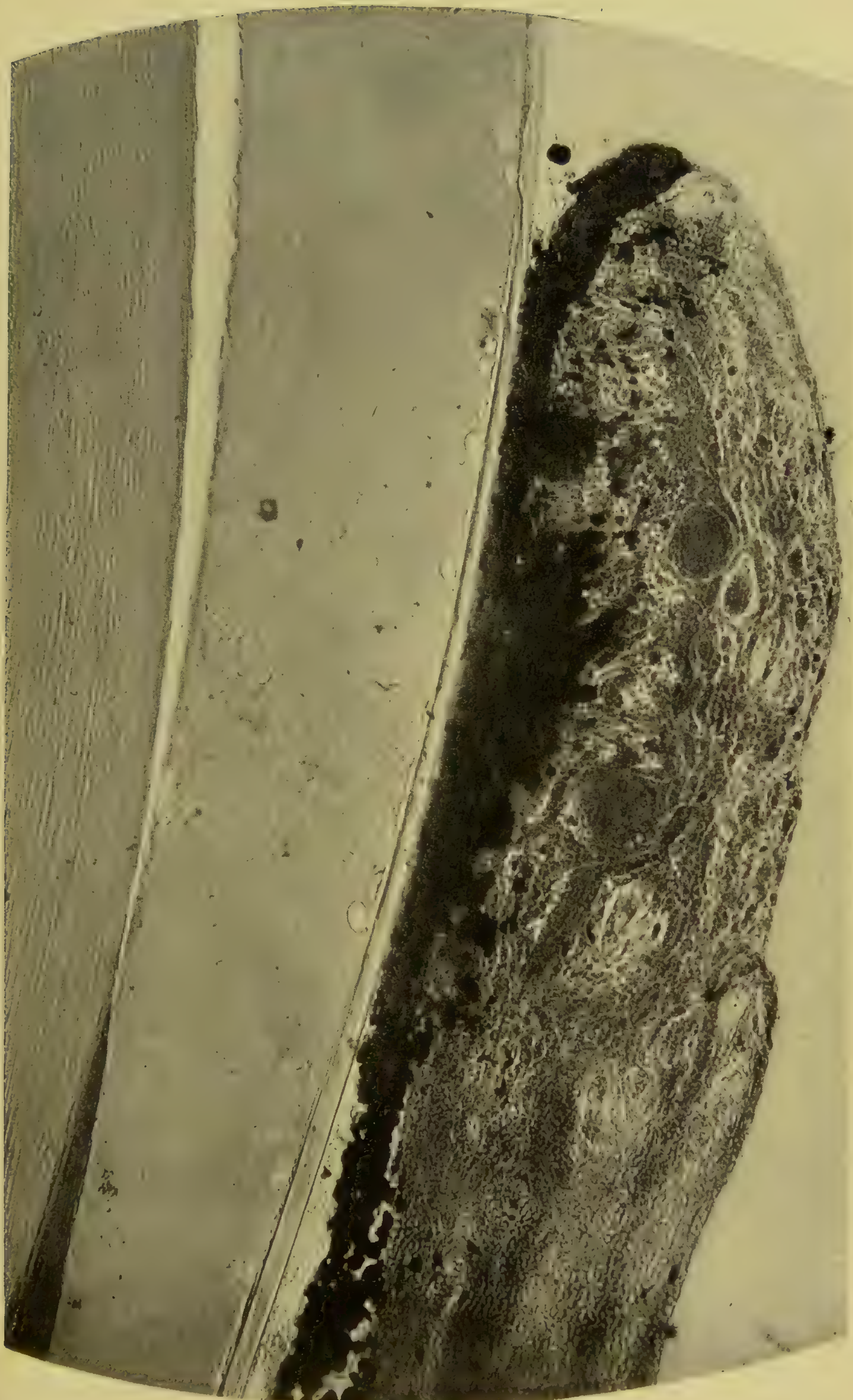
Cataractous degeneration of lens-fibres. (Photographed by Dr. George W. Norris.)

Fig. 33.



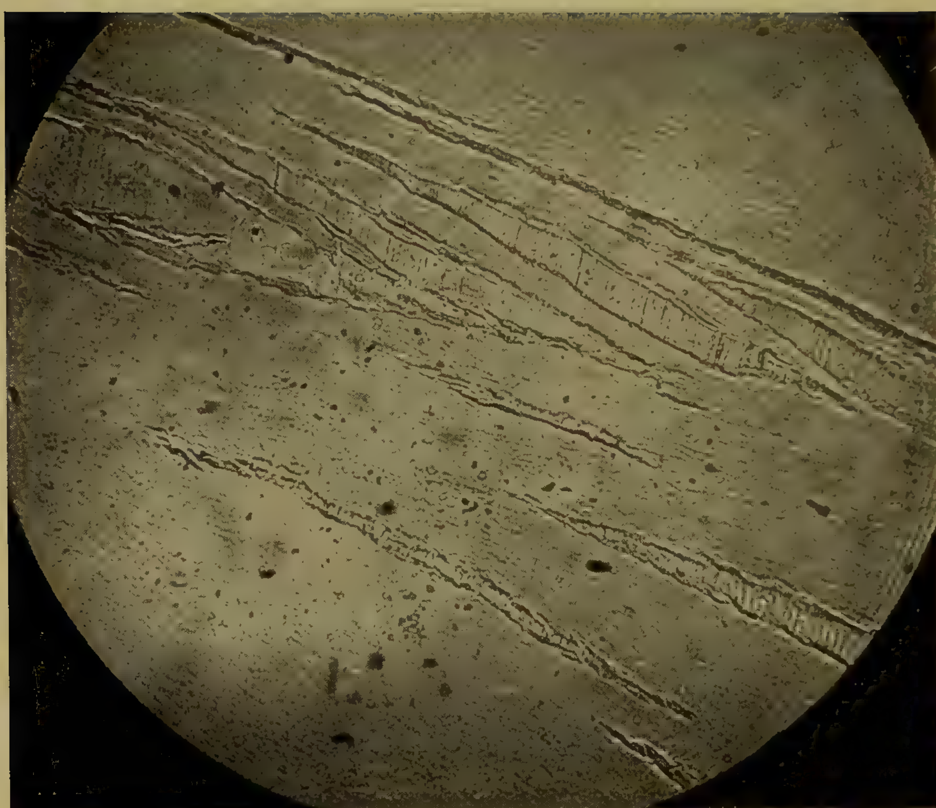
Cataractous degeneration of lens (near the vortex). (Photographed by Dr. James Wallace.)

Fig. 34



Gelatiniform degeneration of lens-fibres beneath a posterior synechia.
(Photographed by Dr. James Wallace.)

Fig. 36.



Spindle-shaped masses with striation transverse to length of spindle.
(Photographed by Dr. James Wallace.)

Fig. 37.

Proliferation of capsular epithelium in an over-ripe cataract.

(Photographed by Dr. James Wallace.)

have been aptly likened by Becker to the appearance of the jointed algæ. In his anatomy of the healthy and diseased lens he has most accurately described and beautifully figured these appearances, as is seen in Fig. 35—a reproduction of one of the plates in his work. Fig. 36, from a photomicrograph of one of my own preparations (Dougherty), exhibits large spindle shaped spaces filled with a granular mass which is coarsely striated in a direction transverse to the length of the spindle.

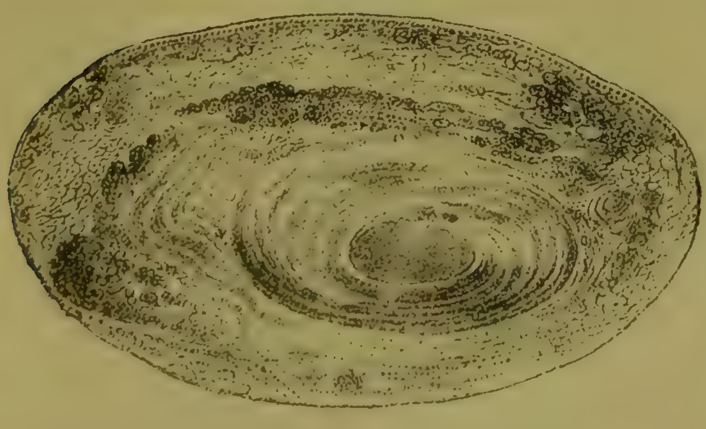
FIG. 35.

Nucleated and swollen lens-fibres, "alga-like" formations in the interfibrillar spaces. (Becker.)

Where the changes continue we have molecular and fatty degeneration of the cortical fibres, which eventually break up into granular detritus, cholesterine crystals, oil globules, calcareous granules, and rounded albuminoid masses (the so-called Morgagnian balls). The molecular and fatty degeneration of the lens-fibres is well shown in Fig. 29.

Fig. 37 shows the structure of one of the little white dots which form just beneath the capsule in over-ripe cataracts. The cells involved in it are evidently products of the capsular epithelium. They have, however, become gigantic in size, their contents are granular, and examination with a higher power shows that in some the nucleus is no longer demonstrable, while in others it is ill defined and breaking down, and in others again it is irregularly oval or irregularly round. Becker has seen in such proliferating epithelial masses at times karyokinetic figures and division of the nuclei.[1] Where the degeneration of the lens has proceeded so far that the more recently formed fibres at the vortex degenerate and give way, we frequently find that the proliferating capsular epithelium gradually pushes its way to the equator and beyond it on to the posterior capsule, and that this portion of the capsule is covered by a single or even a double layer of epithelial cells. Where the cortical fibres entirely degenerate they break down into a whitish semifluid mass, in which the dense and undissolved nucleus floats about with

FIG. 38.



Section of Morgagnian cataract. (Becker.)

[1] Becker, Anatomie der gesunden und kranken Linse, Fig. 9, 10.

the motions of the eye, usually from its greater specific gravity lying at the bottom of the capsular sac. Fig. 38 (after Becker, his Fig. 36, Tab. viii.) shows a successful section of a Morgagnian cataract enveloped in its capsule. The nucleus itself is undergoing degeneration, frayed and splitting into layers surrounded by masses of broken-down lens-fibres and detritus. At the equator of the lens are large masses of proliferating capsular epithelium.

The chemical changes in cataractous lenses and in the nutrient fluids surrounding them, which correspond with the loss of transparency of the fibres, and which precede and accompany their degeneration and breaking down, have been but little studied. E. v. Jaeger[1] tapped the anterior chamber of normal and of cataractous eyes and had the fluid so obtained analyzed by Kletzinsky, and found that the aqueous of cataractous eyes was richer in albumin than that of normal ones. Leber[2] reports that examination of the aqueous in a diabetic patient not only demonstrated the presence of sugar, but also a large quantity of albumin. Deutschmann[3] asserts that during the maturation of cataract the aqueous contains more albumin than does that of normal eyes. I have been unable to find any analyses of the human vitreous. Deutschmann found by careful drying and weighing that the non-cataractous senile lens becomes steadily heavier with age, and that the absolute amount of water diminishes while that of the solids increases. Priestley Smith[4] has proved that the cataractous senile lenses are smaller and weigh less than do healthy lenses at the same time of life, while Becker[5] claims that senile cataractous lenses contain very considerably more water than do unclouded senile lenses. As to the composition of the cataractous lens itself, Cahn[6] found, using as material lenses extracted by Laqueur, that in them the quantity of albuminous material soluble in water and carbonic acid was diminished, and concludes that during life a part of the albuminous material has become insoluble. He also found lecithin in such lenses in quantity above normal. According to Jacobsen (as reported by Zehender and Matthiessen)[7] cholesterin, which appears to exist in small quantities in young and normal lenses, was much more abundant in senile lenses, and still more so in senile cataractous ones, and that in cataracts there was three times as much in the nucleus as existed in the cortical substance. He attributes its presence to the breaking down of albuminous material in the lens, and clinical observation, which at times shows us cholesterin crystals in degenerating lenses, lends strength to his view.

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[1] Von Jaeger, Einstellungen des dioptrischen Apparats, S. 139–148, Wien, 1861.

[2] Leber, Archiv für Ophthalmologie, xxi., 3, S. 327.

[3] Deutschmann, ibidem, xxv., 2, S. 214.

[4] Priestley Smith, loco citato, vide p. 45.

[5] Becker, Anatomie der gesunden und kranken Linse, S. 98.

[6] Cahn, Zur physiol. und pathol. Chemie des Auges, Strassburg, 1881.

[7] Zehender and Matthiessen, Klinische Monatsblätter für Augenheilkunde, xv., S. 237–307.

## CAPSULAR CATARACT.

Besides those forms which have been already discussed as part of the development of senile cataract, we have proliferation of the capsular cells to a great extent whenever there is an extensive inflammatory adhesion of the iris to the anterior capsule, and at times sufficient disturbance of the nutrition of the underlying lens-fibres to cause either partial or total cataract. These changes may be congenital, and caused either from persistent pupillary membrane remaining adherent to the anterior capsule or by inflammatory exudation during the fœtal state. Capsular cataract may also occur from similar inflammatory adhesions of the iris, occurring at any time after birth. The accompanying figures (Figs. 39 and 40) show a partial

FIG. 39.

FIG. 40.

Partial capsular and lenticular cataract caused by retained pupillary membrane. (Norris.)

capsular and lenticular cataract caused by retained pupillary membrane which was adherent to the anterior capsule. The patient was a young girl, aged sixteen, who thought that her sight was weak and came for glasses. Vision in the right eye was normal, in the left 5/15, and with this eye she could read 0.50 type from twelve to twenty-eight centimetres only, while with the normal fellow-eye the range for the same type was from ten to fifty-eight centimetres. Both eyes were hypermetropic, and there were no coarse changes in the eye-ground. Fig. 40 shows the pupillary membrane and opacity as seen by the mirror, and Fig. 39 as seen by oblique light. Careful examination showed that the opacity extended for some distance into the lens-fibres. Although there was a strong reflex from the lens, there were no other opacities. Fig. 41 (copy of Fig. 1, Taf. xi., Becker's Atlas) shows a partial cataract caused by a granuloma of the iris and consequent adhesions between the iris and capsule. There is new-formed fibrous tissue between the iris and anterior capsule. The latter is raised in folds, and at the equator there is a mass of large vesicular lens-cells. Fig. 42 (Becker, Taf. xiv., Fig. 63) shows the appearance of the lens in a complicate cataract from the buphthalmic eye of a seven-year-old child. A false membrane fills the pupil and binds the iris to the capsule of the lens. The an-

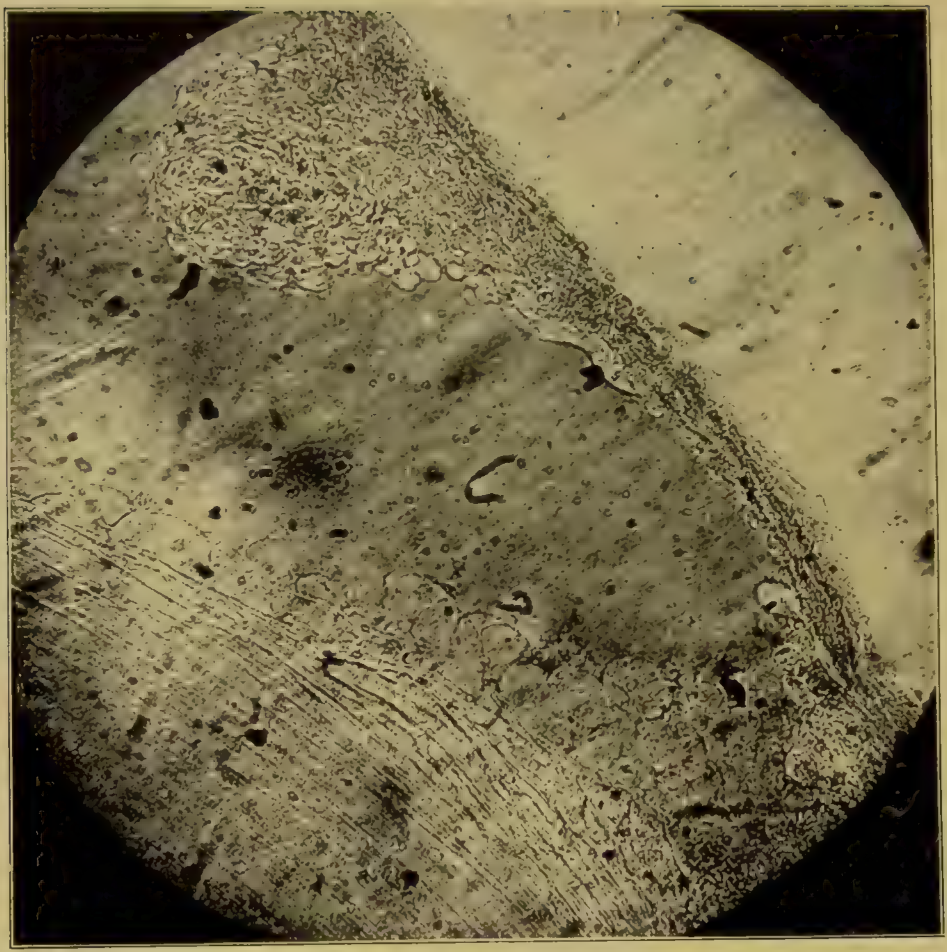
FIG. 41.

Capsular-complicate cataract. (Becker.)

FIG. 42.

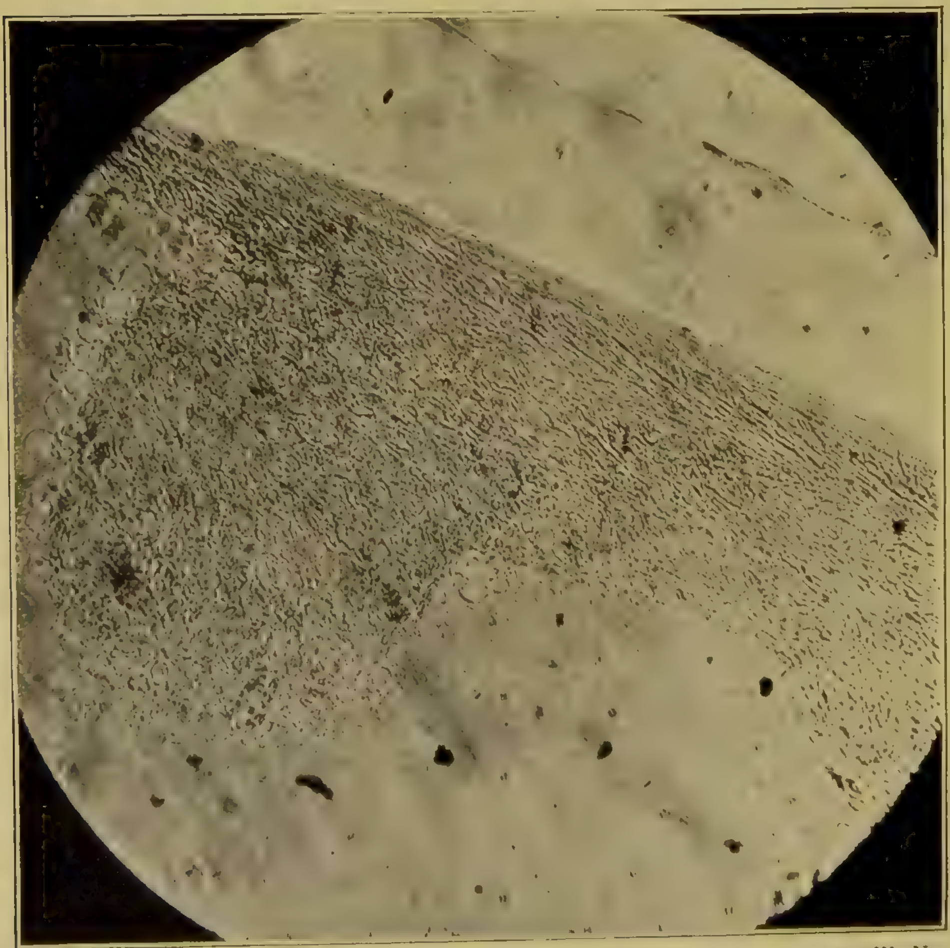
Capsular cataract caused by pressure of a granuloma of the iris. (Becker.)

Fig. 43.



Marked epithelial proliferation in intact capsule. (Photographed by Dr. George W. Norris.)

Fig. 44.



Marked epithelial proliferation in intact capsule. (Photographed by Dr. George W. Norris.)

terior capsule is folded and runs in ridges across the pupillary space. Beneath it is a very large capsular cataract which is covered by a thin layer of capsule with epithelial cells. Behind the capsular cataract lie the lens-fibres, pressed and pushed away from it by an amorphous mass of exudation and degenerative products, while the posterior capsule is covered with a layer of epithelium. Subcapsular epithelial proliferation is usually marked wherever there is a separation of the capsule from the lens-fibres. The accompanying photograph gives a good picture of such proliferation in a lens with absolutely intact capsule, which escaped spontaneously into the conjunctival sac through an ulcer of the cornea, caused by purulent ophthalmia in an infant. Fig. 43 shows a portion of the capsule everywhere covered with a many-layered epithelium. From one corner of the preparation an irregular mound of epithelium projects inward, one side of it rising almost perpendicularly from the capsule to a rounded summit and thence sloping down gradually on the other side to the capsular level. The cells in the centre of the mass are smaller and exhibit an irregularly concentric arrangement, while those of the outer layer are large and vesicular. The outer border of the lens is separated from the capsule by fluid, and the peripheral lens-fibres are irregularly swollen and are separated in places by spindle-shaped spaces filled with granular detritus, while along their outer border stretches a conical mass of vesicular nucleated cells, many layered near the capsule, and dwindling in thickness to a single layer as it recedes from it. Fig. 44 shows the same mound of epithelium more highly magnified, and in it we can see that the older cells at the capsular level have become spindle-shaped and fibroid in character.

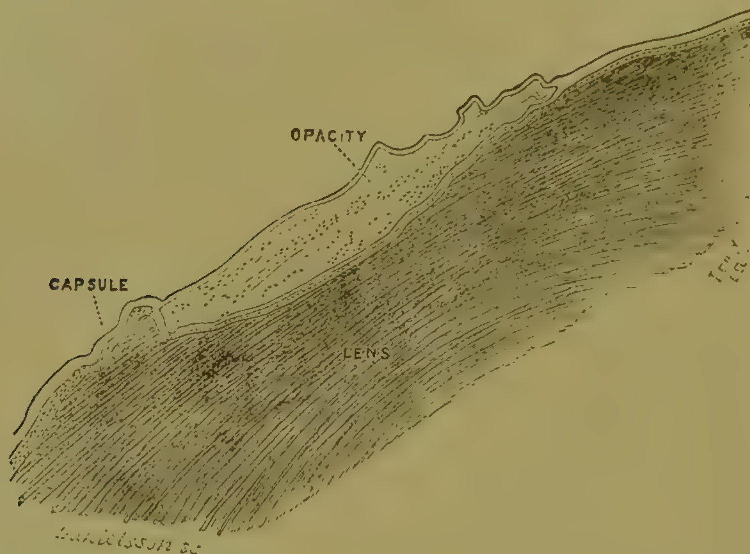
### ACQUIRED ANTERIOR CENTRAL CAPSULAR CATARACT.

The form and appearance of this variety of cataract have already been described under the head of clinical appearances, and reference has been made to the classic observation of Arlt demonstrating its origin. In fact, the subsequent observations of Knies[1] have shown that contact for a few days between the capsule and the edges of a perforating corneal ulcer may suffice to give rise to it. The lenses examined by this author which had escaped from the ulcerated corneæ were apparently perfectly clear, but after hardening, an opaque spot was visible beneath the capsule at the anterior pole, consisting of spindle-shaped cells which gradually passed into the surrounding normal capsular epithelium. E. Treacher Collins[2] has given a considerable number of admirable representations of this form of cataract, two of which are herewith reproduced. One shows a flat form with the anterior capsule folded over it, the capsule splitting at the edge of the growth, and part of it with the epithelium being continued over its posterior surface, which is in contact with the lens-fibres. The other shows a

[1] Knies, Klinische Montsbl für Augenheilkunde, 18–181.
[2] Collins, Trans. Ophthal. Soc. U. K., vol. xii. pp. 89–102.

more pyramidal opacity with a blunt process of newly formed tissue projecting into the lens-substance. This splitting of the capsule is at times seen in capsular cataracts not of the anterior central variety. In such instances it looks as if the posterior newly formed layer of capsule must

FIG. 45.




Acquired anterior central capsular cataract. (E. Treacher Collins.)

have been secreted by the epithelium. Becker, while granting this possible, believes that the epithelial cells exercise a softening influence on the capsules, and by sending their processes farther and farther into it gradually split it into layers, and considers it a real split. He calls attention to the

FIG. 46.


Acquired anterior central capsular cataract. (E. Treacher Collins.)

fact that the inner and outer capsule layers together make just the thickness of the capsule at a short distance peripherally from the capsular cataract, and the first of Treacher Collins' drawings shows also that the two layers about equal the main capsule in thickness. On the other hand, Collins's cases would seem to show that a capsule-like membrane may be

secreted by the capsular epithelium. In the two cases represented, the thinned capsules cover in the growth in front, while the layer of epithelial subcapsular cells seated on a firm capsule-like substance cover it posteriorly and separate it from the lens-fibres. In both these cases the cataract was of several years' standing, but in others which he also pictures, taken from cases examined soon after the formation of the capsular cataract, there was no membrane behind the mass and between it and the lens-fibres, although the layer of epithelial cells leaves the capsule at the border of the new growth and extends some distance behind it. This would look as though the posterior layer of the capsule was developed after a time by the epithelium which had pushed in behind the growth. In some cases the capsular cataract consis'ed of a laminated material, interspersed with degenerating epithelial cells. The studies of Wagenmann and the experiments of Schloesser further confirm this view and make it probable that wounds in delicate vitreous membranes, such as the lens-capsule and the membrane of Descemet, are usually repaired eventually by a new vitreous membrane formed by the epithelial cells beneath a temporary layer of effused and organized fibrin.

## THE FORMATION OF SPINDLE-CELLS IN CAPSULAR CATARACT.

Fig. 47 (after Becker) shows that some of the capsular cataracts also consist of spindle-cells and resemble fibrous tissue. This is often the case, but the resemblance is probably a superficial one, because, as already pointed

FIG. 47.

Subcapsular spindle-cells resembling fibrous tissue. (Becker.)

out by Becker, they develop from epithelial cells, and Schirmer[1] has since demonstrated that a solution of trypsin, which does not at all affect connective tissue, promptly either in acid or alkaline solution dissolves all epithelial productions, and among them the lens-capsule in capsular cataract.

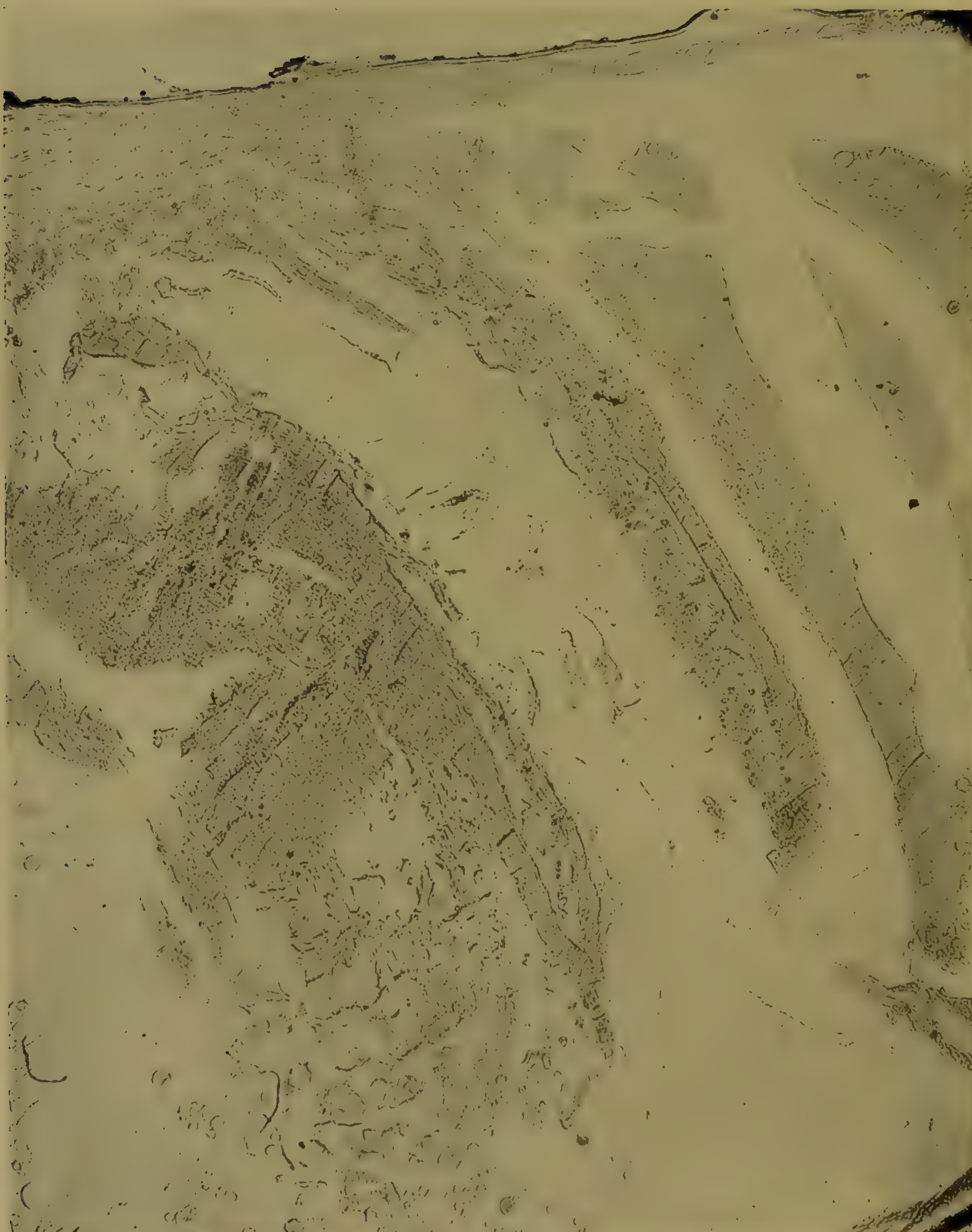
[1] Schirmer, Archiv für Ophthalmologie, xxxv., 3, S. 278.

## SEPARATION AND FOLDING OF THE CAPSULE.

Slight separation of the capsule at the equator of the lens and folds in the anterior capsule may occur from many causes besides that just described under the head of capsular cataract. Topolanski[1] has called our attention to the fact that in some cases of congenital coloboma of the lens the capsule passes over the notch in the same circular line as the capsule elsewhere, leaving a considerable clear and fluid-filled space between itself and the lens. The same fact has been noted by Christen. Topolanski and also Magnus tell us that at times tent-like elevations of the capsule can be seen in perfectly healthy young lenses, due to the drag of the zonula. According to Schoen all simple or senile cataracts commence as a "peripheral capsulitis" and have their starting-point in the separation of the lens-fibres from the capsule at the points where the zone of Zinn is inserted into them. He considers that this state of affairs is caused by strain of the accommodation after the lens has become hard and unyielding, and thinks that similar small detachments of the capsule are caused in the young by the effort to overcome hypermetropia and astigmatism. The initial lesions of cataract according to this author are the separation of the lens-fibres from the capsule and the proliferation of the epithelial cells, which is often followed by a hyaline degeneration and a coagulation necrosis of the cells. Topolanski considers separations of the equatorial capsule (demonstrated through the coloboma of a preliminary iridectomy) to be frequent in all senile cataracts. This author and also Sattler and Inouye consider such separations common in cases of over-ripe cataract with capsular degeneration. Folds in the pupillary space may be seen occasionally after wounds of the eye both penetrating and non-penetrating. Topolanski has observed them caused by the contraction of effused lymph under a posterior synechia after iridectomy, in which case the pull of the cicatrix also made a slight indentation in the periphery of the lens. The folds were few in number when first observed with the ophthalmoscope, but became more numerous from day to day till fourteen were counted. When the eye was examined with a weak-light mirror slight rotations of the latter caused remarkable changes in the appearance of the more or less spindle-shaped elevations of the capsule. With a certain projection of the light they looked dark, and then by slight motion of the mirror lighted up brilliantly like illuminated glass splinters. Inouye[2] also gives an interesting instance of capsular folds in the pupillary space in a case of double traumatic iridodialysis. In this case the folds ran across the pupillary space in a direction at right angles to the direction of the blow producing the injury; while in the spaces above and below, between the periphery of the detached iris and the cornea, the edge of the lens could be seen and was found to be markedly serrated. No trace of capsule or zonula was visible at these points. Larger detachments

[1] Topolanski, Klin. Monatsbl., Maerz, 1897; also Archiv für Ophthal., xli., 3, S. 198.
[2] T. Inouye, Centralblatt für Augenheilkunde, Mai, 1897, S. 147.

Fig. 48.



Traumatic cataract in process of absorption. (Photographed by Dr. James Wallace.)

are seen in many cases where the fluid rapidly accumulates under the capsule, as in the case of diabetic cataract figured by Becker and in the case of the lens of a suppurating eye shown in Fig. 43 and described on page 303. In both these cases there were remarkable ingrowths of the capsular epithelium. In many other instances in over-ripe and Morgagnian cataracts there are considerable spaces between the capsule and the lens, but these, although also separations, are due mainly to shrinking and degeneration of the lens substance with subsequent dissolving and osmosis of the degenerated material rather than to any pull or raising of the capsule.

## TRAUMATIC CATARACT.

1. **Without Rupture of the Capsule.**—Berlin has succeeded in producing cataract by repeated blows on the cornea with an elastic rod. Voelckers[1] has since confirmed these results by producing cataract by tapping an eye with a percussion-hammer or shooting it with projectiles from a toy pistol. Schirmer has shown that contusions of the lens-capsule either cause more active absorption of fluid by the lens or interfere with the excretion of its nutrient fluid, producing an accumulation of liquid between the lens-fibres which pushes them apart. It, moreover, causes granular degeneration of all the fibres with a proliferation of the epithelial cells forming ingrowing masses of tissue, which Schirmer believes to remain transparent and not to give rise to opacities. Clinically, also, we are familiar with cataracts coming on at varying periods after blows on the eye and its vicinity where we are unable to demonstrate any rupture of the capsule.

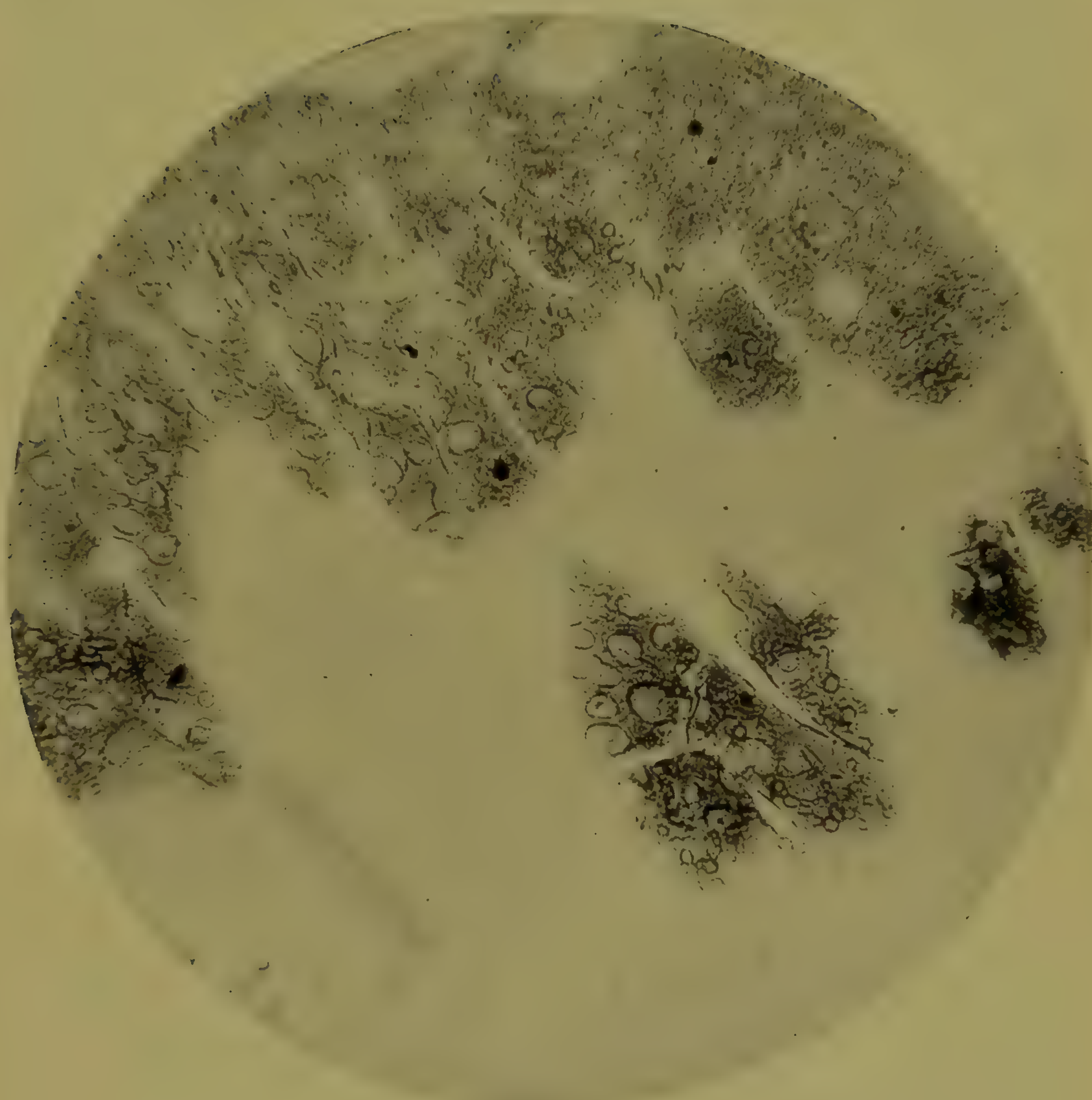
2. **With Rupture of the Capsule.**—As will be shown in the next section, there is good clinical evidence of the healing of small capsular wounds so as to leave no trace of their previous existence. According to Schirmer[2] in such instances a fibrous cap is formed by exudation, and this formation is followed by a proliferation of the capsular epithelium, the cells thus produced secreting a vitreous membrane, which shoves forward between the old capsule and the epithelial layer to cover in the defect. Hoering,[3] who experimented on wounds in the anterior capsule in animals, says that such wounds always tend to unite by simple adhesion, and that failure to do so is usually due to swelling of the lens-fibres. He maintains that the wound always unites if it be made by a sharp instrument, and if it be confined to the capsule without injury to the lens-fibres. The small, irregular masses of lens-matter which protrude through wounds in the capsule soon become opaque from the action of the aqueous and disintegrate, and are dissolved by the further action of the same fluid. Fig. 48 shows a section of a traumatic cataract where the wound has been large and has eventuated in the absorption of the bulk of the lens in the equatorial region. We see broken lens-fibres, frayed at the edges, and between them quantities of irregularly

[1] Voelckers, quoted by Schirmer, Archiv für Ophthalmologie, xxxiv., 1, S. 133.
[2] Schirmer, ibidem, xxxv., 3, S. 220.
[3] Hoering, Ueber den Sitz und die Natur des grauen Staars, Heilbronn, 1844.

round, albuminoid masses without nuclei,—the so-called Morgagnian balls, which are simply the degenerating protoplasm of the fibres. The masses of lens-matter which lie nearest the equator and farther from the wound are best preserved, while those nearer the centre of the photograph, which are closer to the wound and have been more thoroughly exposed to the action of the aqueous, are in a more advanced stage of degeneration, the lens-fibres exhibiting numerous fine transverse striæ and molecular degeneration. Besides the changes above described and pictured, one often finds, as in Fig. 49, which represents another field and a more highly magnified portion of the same lens, curious forms of degeneration, which are seen in the interior of lens-fibres and which themselves still are comparatively intact and preserve their band-like outline. These consist in the formation of large round or ovoid excavations in the fibres, the edges of which are usually sharp cut and which are filled with protoplasmic masses, which are at times nucleated, and which sometimes entirely fill the cavity, or at other times have shrunken away from its walls. These rounded masses often communicate, by prolongations of granular material in the rents in the fibres and in the interfibrillar spaces, with similar masses lying in adjacent cavities. In other instances no appearances resembling nuclei or cells are to be found, but simply clumps of degenerating material. Fig. 50 gives a view of a large part of the shrunken capsular sac in a case of traumatic cataract. Part of the iris is shown in the upper right-hand corner of the picture; between it and the rent in the anterior capsule is seen a mass of inflammatory exudate, partly pigmented. In the interior of the capsular sac lies a small piece of lens-matter broken into fibrils with frayed ends, which are surrounded by a mass consisting of leucocytes, Morgagnian balls, and lens-detritus. It has been frequently observed that in traumatic cataract giant cells are found containing myelin drops and broken-down lens-matter. Lately Wagenmann[1] has called special attention to them, ascribing to them marked histolytic action, and in one case of spontaneous luxation of the lens consequent on iridocyclitis he imputes to these cells an ability to cause absorption of the vitreous capsular material and usure of the capsule. At times we have also a form of degeneration of the lens-fibres, in which they break up into narrow transverse bands, the lines of division being so close as to recall the striation of muscular tissue. As the narrow bands loosen and fall off they become rounded and break up into smaller rounded masses, (*vide* Fig. 51). Wounds of the lens-substance through the anterior capsule make a gray track, by which we can follow their course, and often the only other opacity in addition to this may be radiating opacities in the posterior cortical. Leber informs us that when he removed a piece of the anterior capsule of the rabbit he obtained a nuclear cataract. When the entire lens is absorbed, the anterior and posterior capsule may fold together, stretching across the eye behind the pupil in the level of the insertion of the zone of Zinn and resembling the

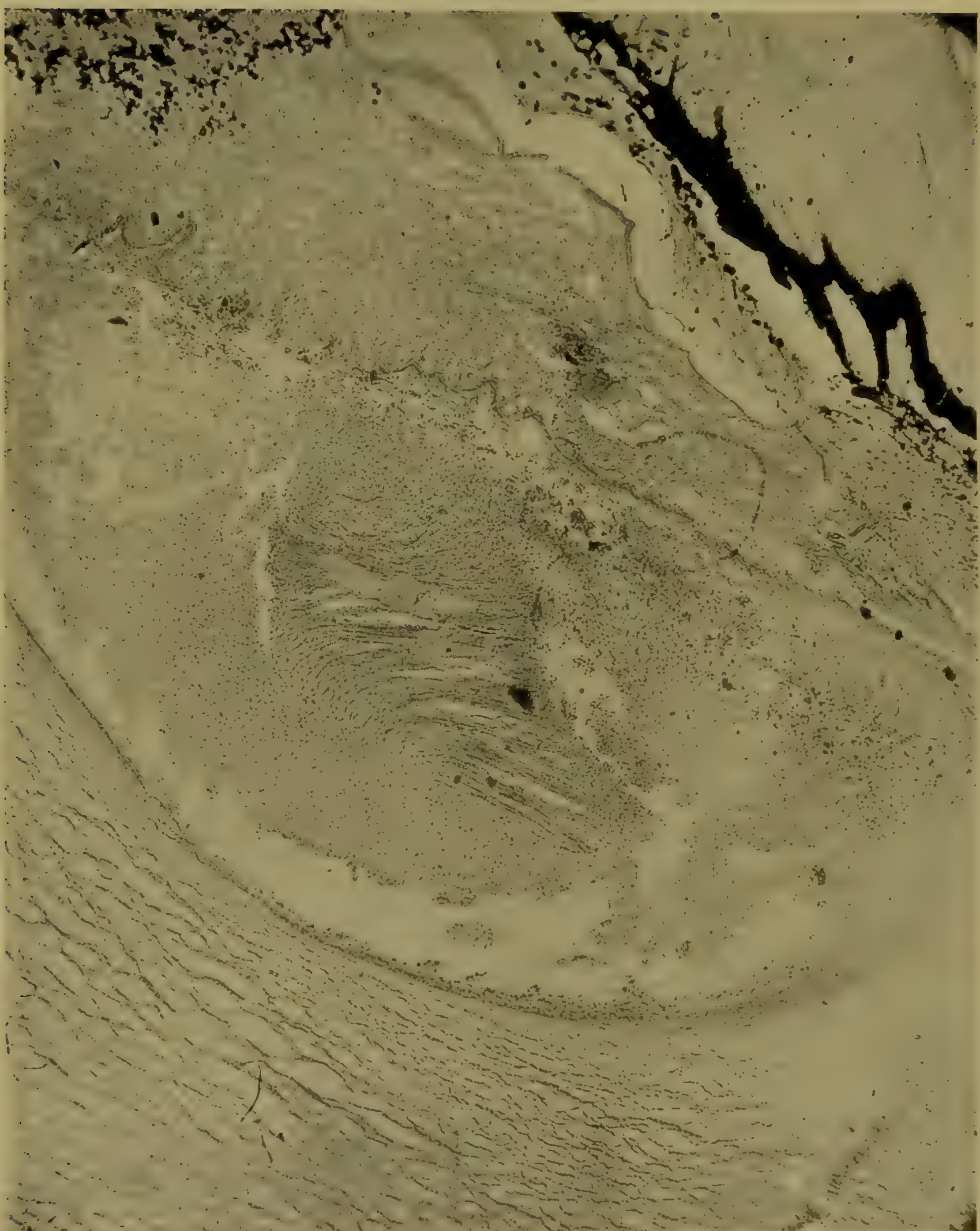
[1] Wagenmann, Archiv für Ophthalmologie, xlii., 2, S. 80.

FIG. 49.



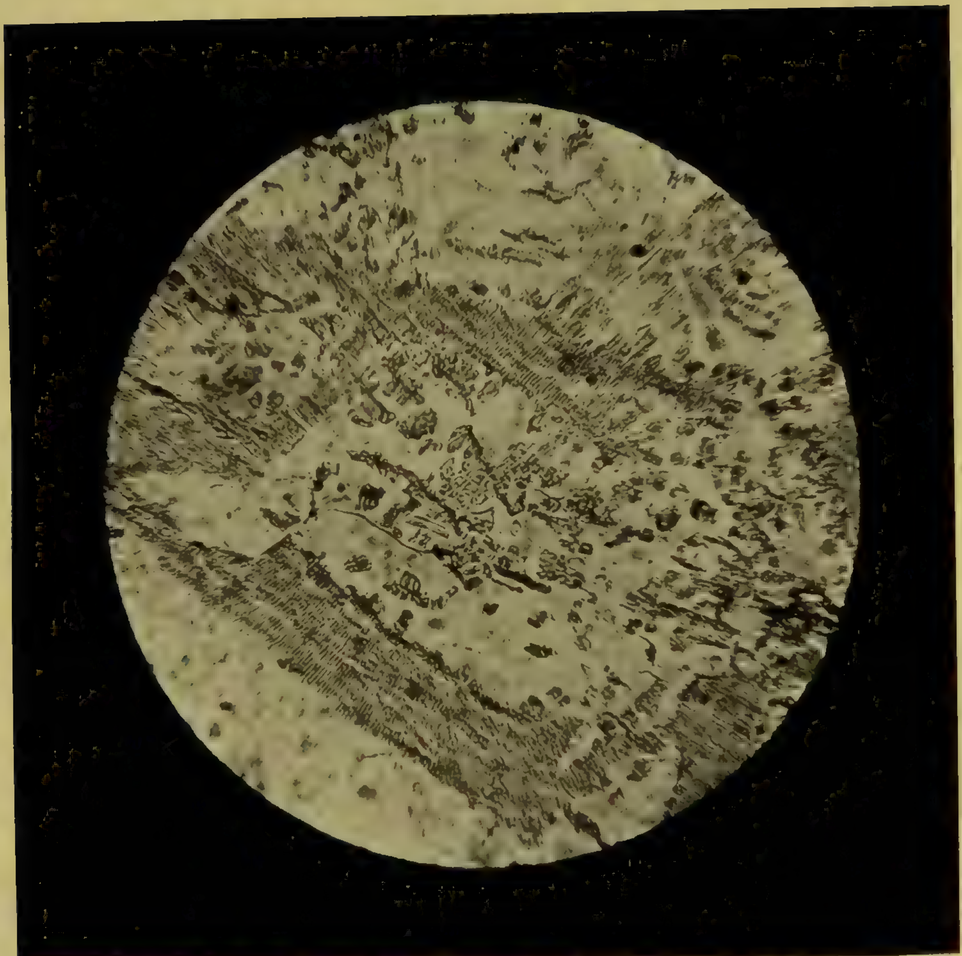
Traumatic cataract in process of absorption. (Photographed by Dr. James Wallace.)

Fig. 50.



Traumatic cataract in process of absorption. (Photographed by Dr. James Wallace.)

Fig. 51.



Traumatic cataract in process of absorption. (Photographed by Dr. George W. Norris.)

secondary cataract resulting from cataract operations. In most instances such complete absorption does not occur, and we have a shrunken lens of irregular shape sitting just behind the pupillary space and often bound by inflammatory adhesions to the cornea or the iris and ciliary body. Violent iridocyclitis, often followed by detachment of the retina and vitreous, or by sympathetic affections of the fellow-eye, only too frequently results.

Of late years a considerable number of cases of traumatic cataract caused by lightning have been recorded, and consequently the experiments of Hess, who produced it by discharges from a Leyden jar, become interesting. The discharges produce contraction of the pupil, chemosis of the conjunctiva of the eyeball, circumscript cloudiness of the cornea, intense hyperæmia of the ciliary body, and clouding of the lens. The opacity of the lens commenced near the equator, then followed clouding of the epithelial cells of the anterior capsule and anterior cortical cataract. Hess[1] likens it to the effect of massage on the lens, and states that in both there is death of the epithelial cells. Silex has reported the case of a child three and a half years old, who was struck by lightning and who remained insensible for five hours. There was no visible wound of the eyes, but both eyes presented pericorneal haze and injection, with corneal and lenticular haze. Eleven months later these conditions had very much diminished but had not entirely disappeared. Leber has collected eighteen cases of injuries of the eyes by lightning. In nine of these cataract formed (exclusive of a tenth case, that of Vossius, where the cataract was due to chorioiditis). The remaining cases displayed the most varied pathological conditions, such as optic neuritis, hemorrhages at the macula, rupture of the chorioid, detachment of the retina, paralysis of the external muscles, opacities of the cornea, and intolerance of light.

In some instances of traumatic cataract accompanied with partial displacement of the lens, the iris is either pushed back or retroflected at the junction of the circular and radiating fibres and partially or entirely disappears from view. The well-known case of von Ammon,[2] where a concussion of the eye was produced by the discharge of a musket loaded with water against the roof of the mouth, is an example of this; while Treacher Collins reports two cases of partially-displaced cataract which were accompanied with rupture of the fibres of the ligamentum pectinatum going to the iris, and by a split in the ciliary body which separated its circular from its longitudinal fibres.

### TRAUMATIC CATARACT PRODUCED BY THE OPERATION OF DISCISSION.

The following photographs show the effect of a careful tentative discission of the lens in an adult of thirty years of age, where, owing to the small amount of resulting absorption, the lens was subsequently extracted.

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[1] Hess, Experimentelles ueber Blitz-Kataract, Bericht der VII. internat. opth. Kongress, Heidelberg, 1888.

[2] Von Ammon, Archiv für Ophthalmologie, i., 2, S. 119.

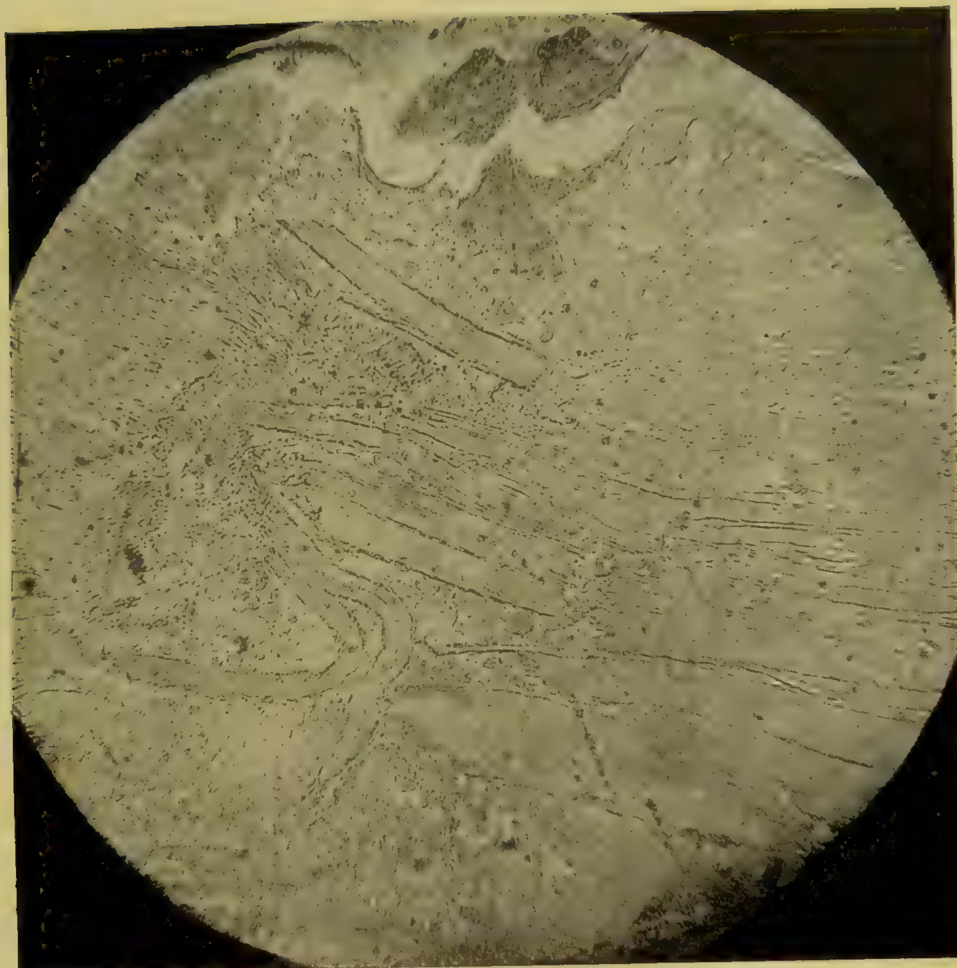
Fig. 52 shows how the lens-tissue has been cut and torn by the lever-like motion of the discission needle, breaking up the lens-fibres into irregular band-like portions with serrated edges at the fracture, and separating groups of fibres, leaving between them spaces filled with granular débris and irregular globules of breaking-down lens-substance. On each side of the capsular rent caused by the entering needle there are evidences of active efforts at repair in the epithelial cells, which have become much enlarged with distinct nuclei and granular contents and are in active proliferation. Fig. 53 shows in a field a short distance from the first how between some of the fibres there are spindle-shaped interstices, in which lie masses of degenerating lens-material, each mass broken up into cylindroid clumps with well-marked lines of division at right angles to the general direction of the spindles. In this instance (Fig. 54) two perforations of the capsule have been made by the needle, lying near to each other. In one of these a band-like mass of lens-fibres fills the opening and protrudes from it. It is frayed and expanded at its outer end, from the action of the aqueous, and is commencing to be covered by a fine layer of fibrinous material. The adjacent opening, which is smaller, has been more completely closed by a heavier layer of similar fibrinous material, as is well shown in Fig. 55, where the nature and extent of the fibrinous exudation which covers the torn ends of the capsule, the breaking-down of the lens tissue, and the large nucleated cells formed by the epithelium are plainly visible. Examination of the fibrinous cap with an immersion-lens shows still more distinctly that its outer side is composed of a very delicate layer of minute spindle-shaped cells with small bodies and long fibre-like processes, all closely felted together. The healing of wounds of the capsule, as might be expected, is closely analogous to the healing of wounds in the membrane of Descemet, where Wagenmann[1] and subsequently Gepner[2] have shown that the cut ends never reunite, but are covered in by fibrous material beneath which a new vitreous membrane is formed by the endothelial cells. Implantation cysts of the iris and ciliary body are sometimes found after traumatic cataract and at times after operation for cataract. The cyst is lined by a laminated epithelium, probably derived from some of the corneal or conjunctival epithelium which has been carried into the wound. In some cases of traumatic cataract we find cilia in the anterior chamber which have been carried in by the instrument causing the wound.

### SECONDARY CAPSULAR CATARACT.

Those forms of capsular thickening and proliferation which occur after the extraction of cataract are good examples of traumatic secondary cataract produced in spite of our endeavors to secure and maintain a clear pupil. Where the lens is not extracted in its capsule, the latter organ is cut or ruptured, and remains behind in the eye. There is always some cataractous

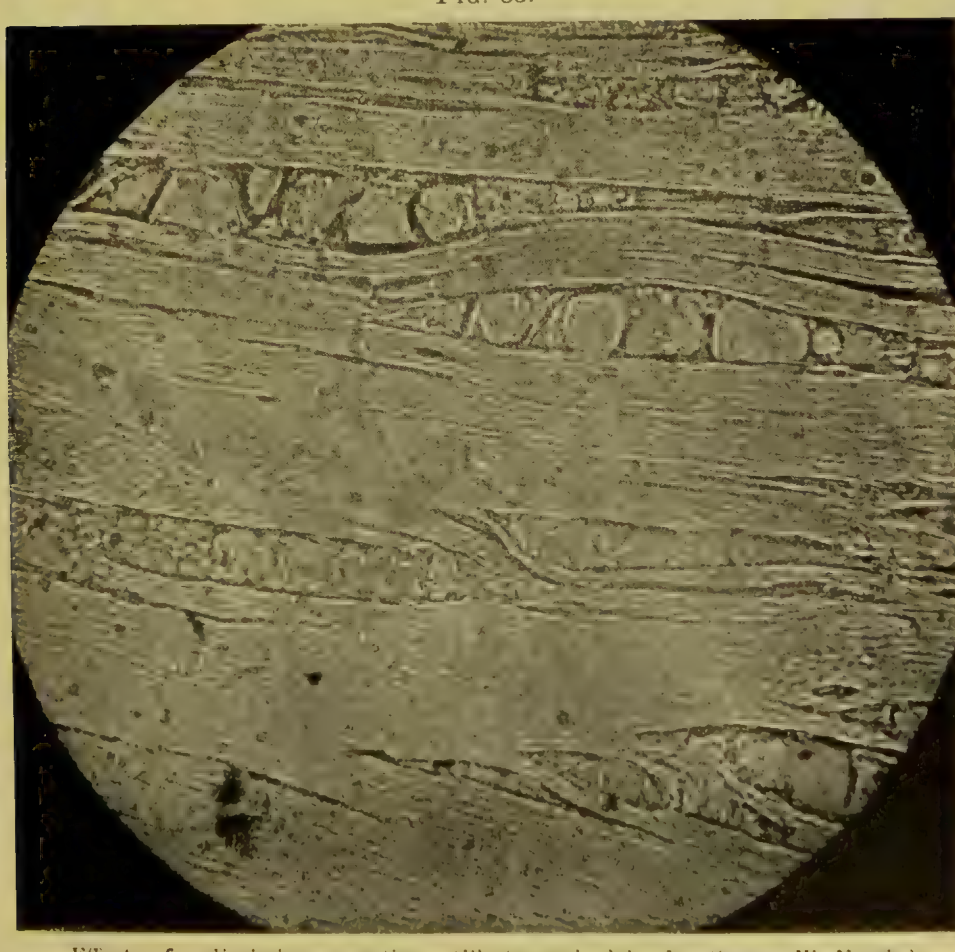
[1] Wagenmann, Archiv für Ophthalmologie, xxxv., 1, S. 172.
[2] Gepner, ibidem, xxxvi., 2, S. 255.

FIG. 52.



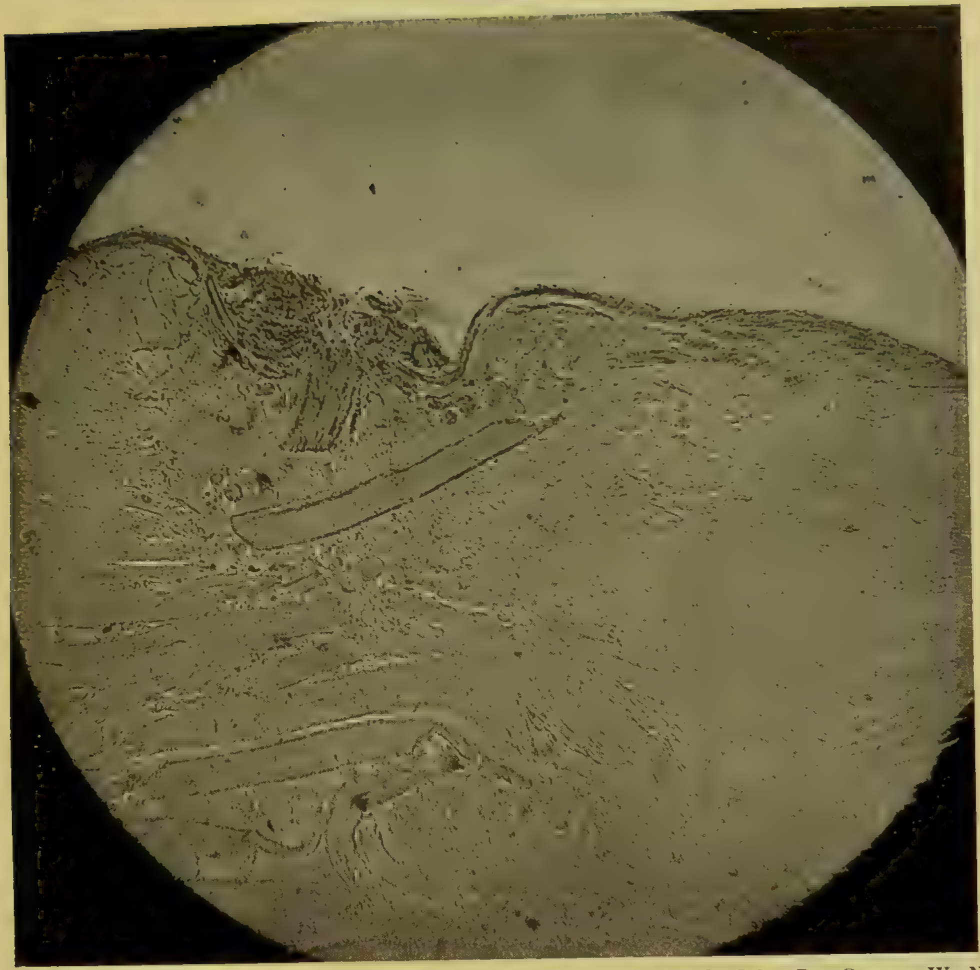
Effects of a discission operation. (Photographed by Dr. George W. Norris.)

FIG. 53.



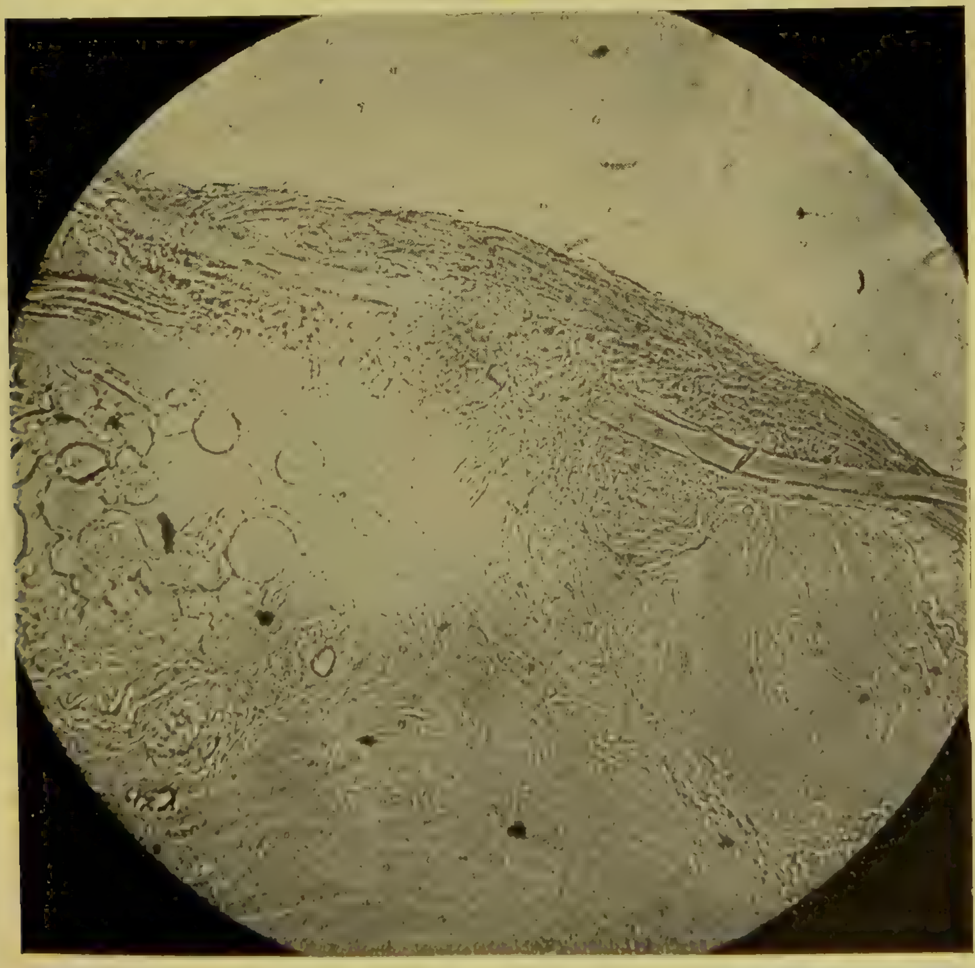
Effects of a discission operation. (Photographed by Dr. George W. Norris.)

Fig. 54.



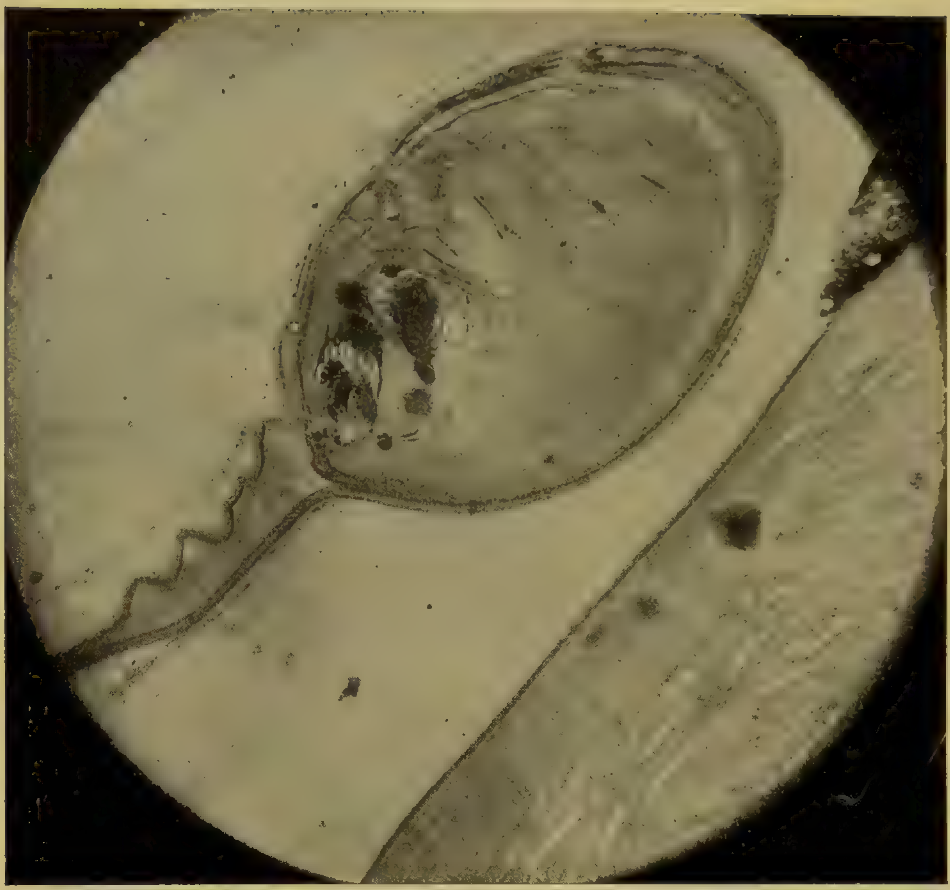
Two perforations in a lens from a discission operation. (Photographed by Dr. George W. Norris.)

Fig. 55.



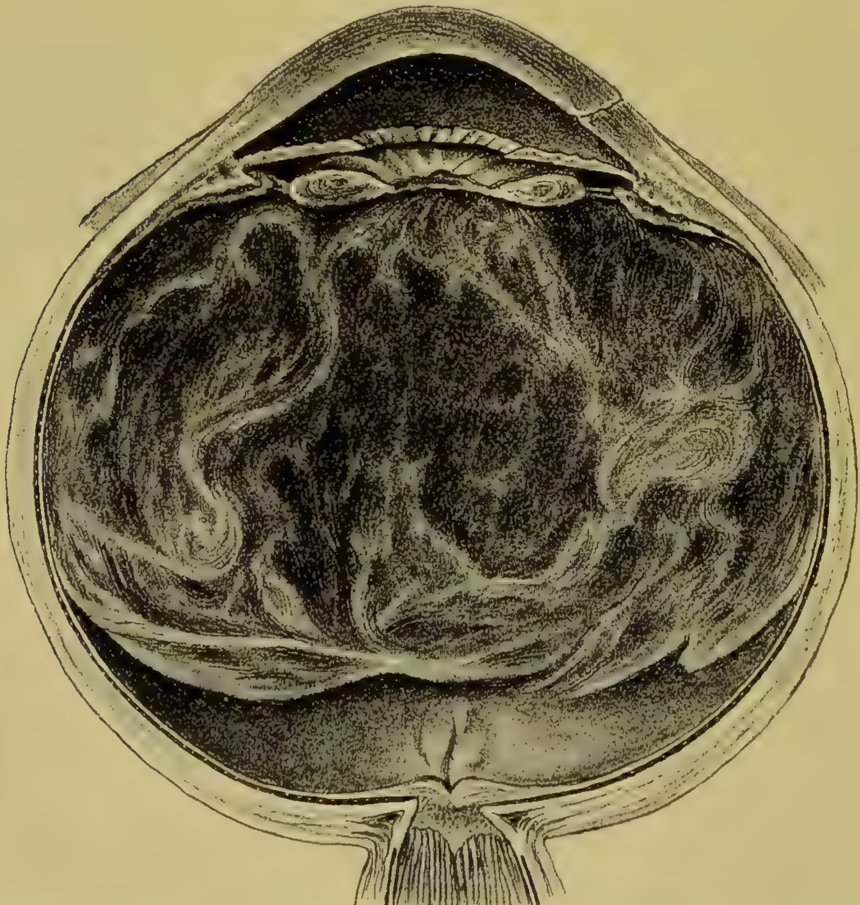
Cap of fibrinous and spindle cells covering the wound made by a discission needle. (Photographed by Dr. George W. Norris.)

Fig. 56.



Proliferation of cells in capsule after extraction. (Photographed by Dr. George W. Norris.)

Fig. 57.



Normal appearance of capsule and enclosed lens remnants after a successful flap operation for cataract. (Becker.)

lens-matter which sticks in the equatorial region. The anterior capsule retracts in folds, and applies itself against the posterior capsule, and, adhesion taking place, the lens matter left behind in the periphery of the capsular sac is shielded from the absorbing action of the aqueous. The capsular epithelium proliferates and produces a transparent mass of vesicular epithelial cells and of abortive lens-fibres which push their way into and around the more or less opaque cortical remnant. The general appearance of such a mass is well shown in Fig. 56. In the young this reproduction of lens-fibres is considerable, and distends the equatorial part of the capsular sac, making a thick ring around the pupil, its position corresponding nearly with the insertion of the zone of Zinn. In young animals where the lens has been removed experimentally, the mass of clear fibres reproduced is so great as to cause Milliot and others to speak of regeneration of the crystalline lens. V. Collucci,[1] Wolff,[2] and later E. Mueller[3] have shown that after extraction of a lens in triton larvæ there occurs a seemingly complete regeneration of it, not by means of any proliferation of capsular epithelium, nor from any ingrowth from the horny layer of ectoderm, but by a growth on the posterior layer of the double layer of epithelium on the iris (pars iridica retinæ), the cells of which, after losing their coloring material, proliferate and form concentric layers, eventuating in a closed sac with transparent fibres resembling in size and appearance those of the original lens.

Where proliferation fills the periphery of the capsular sac and stops at this point, while the pupillary capsule remains clear, we have the normal state of affairs, but in the vast majority of instances we have at various periods after the operation of extraction a clouding of the pupillary capsule, which, when viewed by oblique light, varies in appearance from the finest spider-web to a decided whitish opacity. Fig. 57 gives the normal appearance of the capsule and peripherally enclosed mass after a successful flap-operation for cataract. The exact shape and size of the rent in the capsule are very various, even where an attempt is made to cause a linear cut in it with a sharp instrument. It is readily torn by the instrument, and once opened, the escaping lens often rips and tears it in various directions, so as to present very different appearances in different cases. Fig. 58 shows the appearance of the capsular sac and the position of the rents of the anterior capsule after a simple extraction. The rents in this instance extend fairly out to the zonula. The preparation is

FIG. 58.

Capsular rents after simple extraction. (Hasner.)

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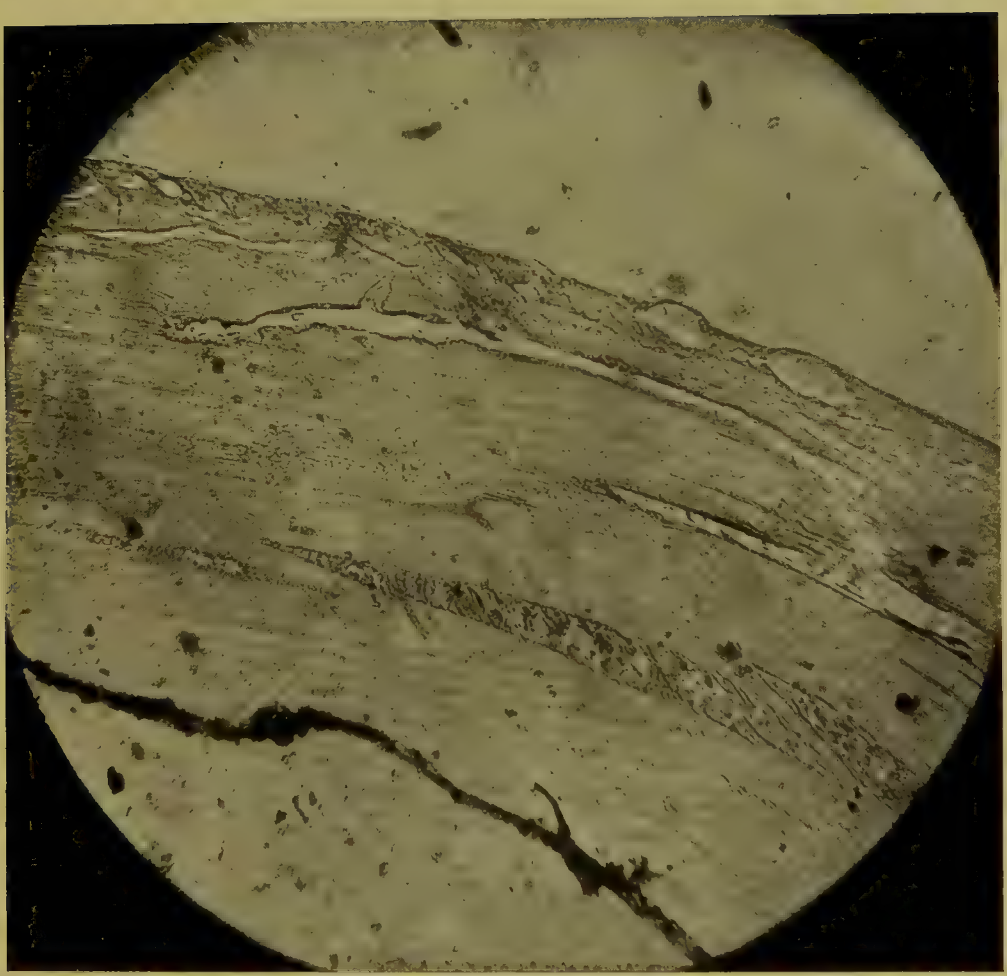
[1] Nagel, Jahresbericht, 1896, S. 34.
[2] Wolff, Biologisches Centralblatt, No. 17, 1894.
[3] E. Mueller, Archiv für micros. Anatomie, xlvii. S. 23.

represented as seen from behind, the eye having been removed from a patient who died of endocarditis fourteen days after the operation. Thickening often takes place in the pupillary capsule without any definite assignable reason, but its occurrence is much favored by any healing of the capsule in the extraction wound or by any ever so slight adhesion to the uveal coat of the iris. The incarceration of a flap of capsule appears to act as an irritant, because it resists the normal contraction of the zonula, which even in lensless (aphakic) eyes takes place in the normal convergence for near work. Healing of the iris or of the hyaloid in the wound often acts in a similar manner and gives rise to recurrent attacks of inflammation. Such impalements also form an inclined plane for infectious material or inflammatory products to pass to the pupillary region. Where blood settles on the capsule it leaves a fibrous coating after its absorption, while attacks of iritis by plastic effusion add to its density and toughness. The drag of the contracting plastic effusion and the effects of ciliary inflammation spreading to the retina and chorioid cause chains of pathological effects which give rise on the one hand to detachment of the retina, to chorioiditis, and to those forms of disease which lead to the shrinking of the eyeball to a sightless stump, and, on the other hand, where they pull forward the root of the iris and the ciliary processes so as to close the angle of the anterior chamber, to increase of pressure and glaucoma.

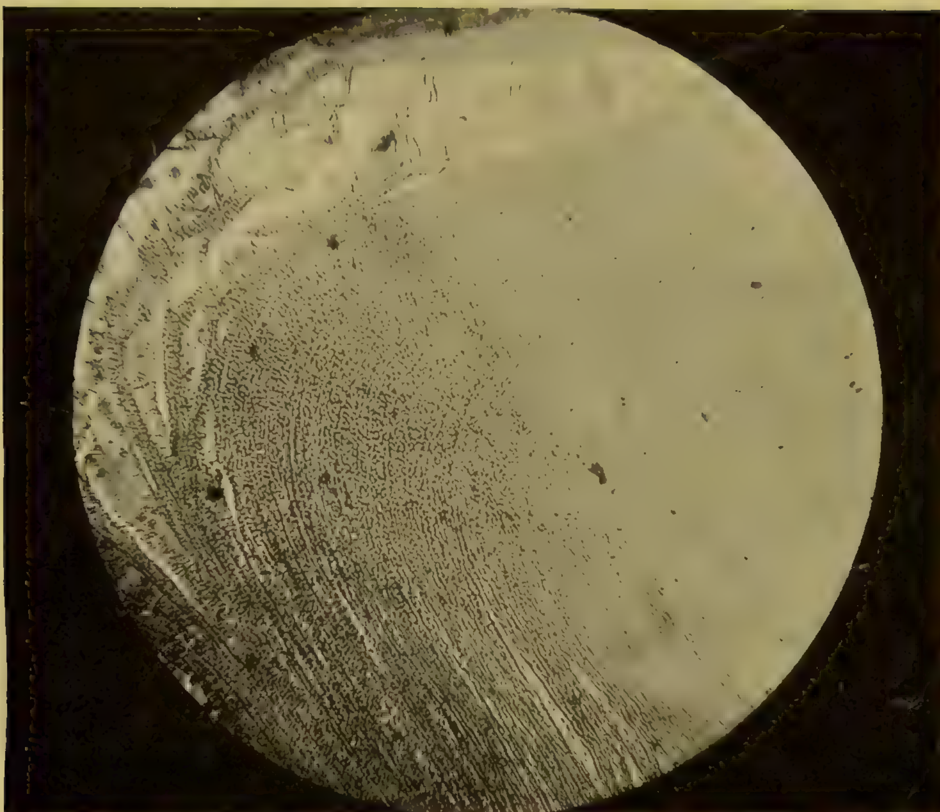
**Secondary Cataract.**—Where the opacities of the lens follow and are evidently due to other preceding diseases of the eye, we have a variety of cataract which is apt to form rapidly. The frequent occurrence of cataract after adhesions of the iris to the lens has been already discussed, but cataract often follows iritis, with clouding of the aqueous and effusion into the anterior chamber when no adhesions have taken place. The nutritive and glaucomatous changes produced in eyes by the growth of intra-ocular tumors often also cause cataract. The accompanying illustration (Fig. 59) represents a cataractous lens taken from an eye which during life showed a detachment of the retina in the lower outer equatorial region, followed by a rapidly-forming cataract which was whitish-gray in appearance. The sections showed a sarcomatous growth under the detached retina. There were no synechiæ or effusions of plastic material into the anterior chamber. The opacity of the lens was greatest in the subcapsular and superficial layers of the cortical, and consisted, as shown in the figure, of various forms of degeneration of epithelial cells, granular clouding of the lens-fibres, with many chinks between the fibres, the larger ones being spindle shaped and filled with clumps of degenerative material and large vesicular cells. Cataract is common also after disease of the chorioid and of the pigment epithelium of the retina, in the latter cases usually commencing as a posterior cortical cataract (*e.g.*, in typical pigment degeneration of the retina). When suppuration of the chorioid occurs, and the vitreous is infiltrated with pus, the lens often becomes softened and opaque. The earlier writers describe the appearance of pus within the capsular sac, mostly between the

Fig. 59.



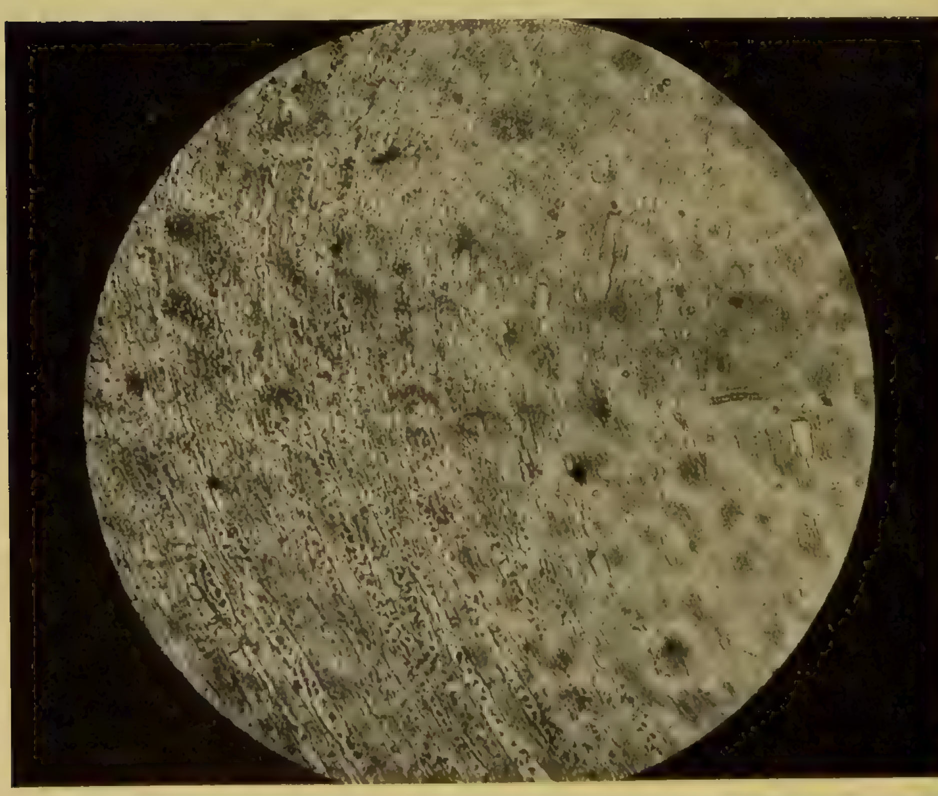
Cataract following tumor of the chorioid with detachment of the retina, the greatest changes being near the anterior capsule. (Photographed by Dr. George W. Norris.)

Fig. 60.



Leucocytes between the lens-fibres in a case of traumatic cataract.
(Photographed by Dr. George W. Norris.)

Fig. 61.



Leucocytes between the lens-fibres in a case of traumatic cataract.—More highly magnified.
(Photographed by Dr. George W. Norris.)

lens-fibres. Thus Weber[1] remarks that he has seen pus-formation within the capsule in cases of artificially produced abscess of the vitreous, due to proliferation of the capsular epithelium, while Moers[2] is of the same opinion, and describes and pictures the endogenous formation of nuclei in the lens epithelium and fibres. Knapp,[3] in a case of metastatic chorioiditis, found pus in a lens with intact capsule. The anterior epithelium was single-layered and intact, the equatorial epithelium proliferating, and many of the lens-fibres swollen into a spindle form. In some, several such swellings were close to each other, each with a nucleus and nucleolus. Goldzieher and Becker also saw pus within the capsule, but attribute its presence to an in-wandering of leucocytes, made possible by thinning and usure of the capsule, as was proved by the actual measurements made by Becker. In all probability most of the earlier observation of leucocytes within the capsule when there was no perforation or usure of this membrane were cases in which proliferating masses of epithelial cells had been mistaken for them. Figs. 60 and 61 show quantities of leucocytes in lines pushing their way in the cracks in the fibre-layer in a case of traumatic cataract. The leucocytes have evidently entered from the anterior chamber through the ruptured capsule and lie in interstices between the fibre bundles, which themselves show granular degeneration. On the other hand, Figs. 43 and 44 show masses of proliferating epithelial cells in a case of unbroken capsule, where the lens, freed from its zonular attachment by the inflammatory processes of purulent ophthalmia, floated out into the conjunctival sac through a perforating ulcer of the cornea. The lens-fibres have been freed from their attachment to the capsule, and between the two is a fluid-filled space, into which project irregular rounded masses of epithelial cells, which, where they are small and round, could readily be mistaken for leucocytes. Most of the more recent writers deny any considerable proliferation of either the lens-fibres or of the capsular epithelium. Thus Iwanoff "never saw nuclear division in a fully developed lens-fibre, but could always convince himself that where there were apparently two nuclei in one lens-fibre there were really two fibres matted together." Deutschmann[4] found usure of the anterior and posterior capsules with leucocytes sticking in the capsule.

### CALCIFICATION AND OSSIFICATION OF THE LENS.

In lenses which have long been cataractous, especially in those where the opacity has developed as a sequence of chorioidal disease, we often have granular deposits of phosphate and carbonate of lime within the capsule, which at times, especially in young subjects, goes on to complete calcification of the lens. According to Stellwag[5] such deposits of lime salts are

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[1] C. O. Weber, Virchow, Archiv, xix. S. 367.
[2] Moers, ibidem, xxx. S. 45.
[3] Knapp, Archiv für Ophthalmologie, 1867, xiii., 1, S. 127.
[4] Deutschmann, ibidem, xxvi., 1, S. 135.
[5] Stellwag, Lehrbuch der Augenheilkunde, S. 668.

sometimes in the crystalline form. Usually the infiltration commences immediately beneath the capsule, as in a case reported by Middleton Michel, where the capsule and adjoining portions were calcareous. On incision of this shell, the nucleus was evacuated, while the capsule and adjacent cortical were so stiff that they still maintained the curvature of the lens. Any approach to complete calcification of the lens is always a slow process and generally occurs in eyes which have been diseased for years. Wagner, in 1851, was the first to report having found true bone with Haversian canals and bone-corpuscles in the shrunken lens of a shrunken eyeball. Most authors, however, doubt the possibility of bone formation from the cells of epithelial origin which constitute the lens, believing that it only forms in fibrous tissue, and explain the occurrence of bone in this situation either by rupture of the posterior capsule and the entrance of vitreous humor, which may in some instances carry newly formed vessels, or (Becker) that in shrunken eyes a vitreous formation may be pushed nearly into the place once occupied by the lens. Voorhies[1] describes true bone formation in the lens, Keyser[2] reports a case of formation of bone in the lens, and Alt has seen bone within the capsule in two cases, but thinks that it was preceded by usure of the capsule and the formation of fibrous tissue.

### THE FORMATION OF CRYSTALS IN THE LENS.

In the preceding paragraph the occasional occurrence of *crystals* of the carbonate and of the phosphate of lime have been referred to. More common, although rare, is the formation of cholesterine and tyrosine crystals in the lens. As previously mentioned, cholesterine is to be found in every lens, in greater abundance in the lenses of the old than in those of the young, but it is seldom that it crystallizes in demonstrable quantities in this organ. Crystalline deposits of it are more common in the vitreous, and attention was first called to them by Parfait Landrau in 1845. Since the invention of the ophthalmoscope the occurrence of synchisis scintillans has occasionally been seen by all ophthalmic surgeons of considerable clinical experience. A deposit of cholestèrine crystals may also occur on the cornea, and the author has recently seen crystals of it thickly sprinkled on the membrane of Descemet and in the posterior layers of the cornea in a case of Dr. Ring's[3] and also in one reported by Dr. Charles A. Oliver.[4] In the lens the presence of cholesterine crystals has several times been proved by the extraction of that organ and microscopic examination of it. It may appear as a rarity either in the early or in the last stages of cataract, although it is much more frequent in the latter. The first record of cholesterine in the

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[1] Voorhies, Archiv für Augen- u. Ohrenheilkunde, vii., 2, S. 811.

[2] Keyser, Report of Fifth International Ophthalmic Congress, 1876, p. 131.

[3] G. Oram Ring, Ophthalmic Section of the College of Physicians of Philadelphia, February, 1898.

[4] Charles A. Oliver, ibidem, October, 1898.

lens was probably that of Adam Schmidt,[1] who found it in the diseased eye of a peasant girl, aged twenty-five, which, on pressure, felt as soft as "an over-ripe fig." In the anterior part of the cataract there was a "glittering red, silvery, and golden powder," with a deposit one line deep of the same material at the bottom of the anterior chamber, which would disperse itself through the aqueous with every sudden motion of the head. On extracting the cataract there was an escape of aqueous, filled with glittering crystals, as well as of some fluid vitreous. Crystals of cholesterine may also occur in lenses in healthier eyes which are still sufficiently transparent to allow of some useful vision. Krautschneider[2] found them in the deeper layers of a lens in an eye with V. = 6/60. Such crystals may remain in the lens a long time without apparent change. Thus, Tweedy[3] saw them remain unchanged for months and Lang for ten years. Usually, however, the lens in which they are deposited undergoes further degeneration, and they float in the *débris* within the capsular sac. Von Graefe records an interesting case in which the crystals were deposited in the anterior and posterior cortical of a still partially transparent lens, and in which he could follow the breaking down of these layers until the nucleus floated freely in the liquefied and crystal-charged material within the capsule. The author has encountered two marked cases of cholesterine deposit in the lens, one in a soft and partially atrophic eye, where the anterior cortical was filled with it, and in which there were also masses of crystals lying at the bottom of the anterior chamber. In the other, in a partially formed cataract of a negress of fifty years of age, who still had a vision of 5/40, there were quantities of cholesterine plates in and between the lens fibres of the anterior and posterior cortical. The plates were arranged at various angles, and when lighted up by oblique light many of them were struck at the polarizing angle and presented a most gorgeous display of prismatic red and green, such as one sees in similar crystals when laid on a slide and viewed with the polarizing microscope, while the crystals standing at other angles appeared grayish white. In the lens of the fellow-eye there were minute angular gray spots, probably cholesterine crystals in the process of formation. It is stated that where we have within the capsule of a degenerated and semifluid lens such accumulations of phosphate of lime or of cholesterine, if an operation is attempted the lens should be extracted in its capsule, as the crystals do not all escape, and those lying in the iris in either the anterior or posterior chamber are said to prove very irritating to the eye.

### INFLUENCE OF SYPHILIS.

The effects of syphilis on the lens are usually indirect, by affecting the blood-vessels of the eyeball or (according to Michel) the carotids without

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[1] Schmidt, Die Cataracta (aus den hinterlassenen Papieren) von J. A. Schmidt in Wien mitgetheilt von Herrn Dr. Eble in Wien, Von Ammon's Zeitschrift für Ophthalmologie, 1831.

[2] Krautschneider, Beiträge zur Augenheilkunde, Heft xxvi. S. 34.

[3] Tweedy, Lancet, 1874, ii. p. 529.

disease of the intermediate vessels. It may, however, be infiltrated by adjacent gummatous growths, as in the case reported by Scherl.[1] A gumma of the iris and ciliary body destroyed part of the anterior leaflet of the zone of Zinn, and a small-celled infiltration extended through Petit's canal and inserted itself in many places to varying distances between the fibres of the lens. In a similar manner gliomatous growths may after perforation of the capsule push their way between the fibres and thus infiltrate the lens.

## BLACK CATARACT.

*Cataracta nigra* is a name under which are included two varieties of cataract. In the one the lens is blood-infiltrated and blood-stained. In the other we have a lens where the cortical has been slowly and completely converted into nuclear tissue which has itself subsequently undergone slight degeneration. Of the first variety von Graefe[2] gives us an instructive history. It occurred in the eye of a youth who had received a severe contusion of the globe. The cataract *in situ* appeared yellow-brown; the lens-fibres were infiltrated with brown coloring-matter, which chemical examination showed to be identical with blood-coloring matter. Gillet de Grandmont[3] also reports a case of black cataract where the presence of blood-coloring material was proved by spectroscopic examination, although he failed to find the characteristic appearances of hæmatin, obtaining only what he characterizes as the spectrum of altered or pigmentary hæmatin. The recent experiments of Linde[4] are interesting as showing the effect of extensive hemorrhages into the anterior and posterior chambers on the entrance of blood-coloring matter into the lens. After such hemorrhages in a rabbit there was a deposit of fibrin on the anterior capsule with enclosed blackish-brown blood-crystals. The layers of the anterior and posterior cortical were stained reddish brown by the dissolved coloring-matter of the blood, the nucleus remaining nearly free from it. McHardy[5] reports a carefully-examined and interesting case which hardly appears to belong to either variety. The lens appeared as "black as charcoal" in a very thick capsule. Spectroscopic analysis by MacMunn showed that it contained no trace of hæmoglobin, of methæmoglobin, or hæmatin, and that therefore the coloring-matter was not derived from the blood, but appeared to belong to the same class of pigments as melanin. Moauro[6] has examined ten black cataracts, and believes that the coloring-matter is always derived from the blood. In his cases it consisted either of hæmatin, hæmatoidin, or melanin. The few cases which have come under the author's observation have all been examples of the second variety; the color has not been charcoal-black, but by transmitted

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[1] Scherl, Archiv für Augenheilkunde, 1892, S 287 (German edit.).

[2] Von Graefe, Archiv für Ophthalmologie, i., 1, S. 133.

[3] Gillet de Grandmont, Archives d'Ophth., May, 1893, p. 279.

[4] Linde, Centralblatt für prakt. Augenheilkunde, Juli, 1896, S. 196.

[5] McHardy, Trans. Ophthal. Soc. U. K., ii. 10.

[6] Moauro, quoted in Centralblatt für prakt. Augenheilkunde, 1896, S. 691; Riforma Med., 1896, ii. p. 818.

light of a rich mahogany-brown. In these cases, as in those studied by Becker, the growth of the lens had gone on uninterruptedly until advanced age, when the germ or formative force of the anterior epithelium had become exhausted and no new fibres were formed; then those last formed, instead of softening and breaking down, applied themselves to the nucleus, and underwent the same hardening process as the earlier-formed nuclear fibres.

## OF THE EFFECT OF HEAT AND COLD ON THE TRANSPARENCY OF THE LENS.

It has been repeatedly demonstrated that the lens of vertebrate embryos and of young animals becomes cloudy after death. Kunde (1857) showed that frogs exposed to severe cold became cataractous, and that the lens cleared when by exposure to heat the animal resumed its natural condition. Michel has shown that this opacity is due to the formation of vast numbers of small, round drops with a high index of refraction in the nuclear fibres of the lens, and that the opacity clears up and disappears at a temperature of from 15° to 20° Centigrade. On cooling, the opacity again develops, but it can again be cleared up by raising the temperature. The same author has demonstrated that a great reduction of temperature in living animals is accompanied by the formation of cataract. Wernek showed that concentration of the sun's rays by a burning-glass caused the lens to split in the direction of its meridians and to become opaque, while more recently Widmark has shown that probably other influences in concentrated light come into play, and that the ultra-violet rays cause a splitting of the lens, accompanied by marked fluorescence; and Beer, Walther, Mackenzie, Arlt, and Hasner, from their clinical experience, all adduce radiant heat as a predisposing cause of lenticular opacity. Meyhoefer,[1] after an examination of five hundred glass-blowers, found in fifty-nine cataract in various stages, mostly on the side exposed to the heat (left side), and accompanied by a characteristic discoloration of the skin of the face on the same side. He attributes these appearances to the enormous sweating and local abstraction of water from this side of the body. His statistics, however, do not show a very high percentage of cataract. Taking four hundred and forty-two of the above cases under forty-two years of age, only forty-two—that is, 9.5 per cent.—showed lens opacities, a ratio which would appear to be rather below the normal, since Schoen, out of four thousand four hundred and eighty-three cases under forty years of age, taken as they came to the Leipzig clinic, found 11 per cent. with traces of cataract. Hirschberg[2] reports that out of thirty glass-blowers in one factory five were affected with cataract and had the characteristic discoloration of the skin of the face. He holds that when an individual is long exposed to high heat, so much is absorbed by the lens as to cause fine internal changes and subsequently loss of transparency. The examination of a considerable

[1] Meyhoefer, Klinische Monatsblätter für Augenheilkunde, 1886, S. 49.
[2] Hirschberg, Centralblatt für Augenheilkunde, April, 1898, S. 118.

number of cases of men who were constantly exposed to intense radiant heat (puddlers, stokers, glass-blowers, etc.) has convinced the author that such exposure, if long continued, does result in a disturbance of the nutrition of the eye. The ophthalmoscope shows that in men long pursuing such trades the head of the optic nerve is markedly over-capillary, and that the reflex from the chorioid is of a far more intense red than usual. The retinal epithelium is disturbed, and appears woolly and granular, and in a large proportion of such cases we find greater or less opacities in the lens. Von Ammon[1] has studied the effects of heat on the eyes of persons who had been burned to death in an accident on the Versailles Railway. One-half of the body examined was almost entirely carbonized; on the other side, which was less burnt, the eye was removed from the socket. There was slight wrinkling of the sclerotic and of the upper part of the cornea. The retina was detached, the vitreous shrunken, resembling in parts coagulated albumin; the capsule of the lens was white and thickened, and the lens-substance itself was white, opaque, and in places closely adherent to the capsule.

### EFFECTS OF MASSAGE ON THE LENS.

Foerster, who first called our attention to massage as a means of ripening cataract, appears to have thought at first that it only hastened the degeneration of lens-fibres in cases where the cataract had already commenced. Both Hess and Schirmer have shown us, by means of experiments on animals, its effect on normal lenses. The latter opened the anterior chamber, and, without making any iridectomy, rubbed the lens through the cornea with a strabismus-hook. By this procedure out of fifty-two trials he produced more or less opacity forty-six times, while in six cases he was unable to detect any lenticular change. Slight rubbing produced a fine radial striation of the superficial fibres, which increased in size and intensity for a few hours until it occupied a space corresponding to the dilated pupil. This cleared up to a certain extent, but never completely, and three months afterwards presented the same appearance it had in the second week. There were also deeper-lying opacities, which caused the anterior sectors of the lens to become unduly visible. In ten cases total cataract followed, and he found that this could always be produced by prolonged rubbing (from three to four minutes). Sections of the lens examined by the microscope showed swelling of the cells of the anterior capsular epithelium with vacuoles in their protoplasm and nucleus, with gradual death and disintegration of the latter. The lens-fibres themselves became granular and slightly swollen, with separation of their club-shaped extremities from the capsule, the resulting space being filled with granular material. There were also deeper-lying spindle-shaped spaces between the fibres filled with a similar material. Fig. 62 gives a reproduction of a photomicrograph in a case of unripe cataract where, after iridectomy, the

[1] Von Ammon, Annales d'Oculistique, xxvii. p. 41.

Fig. 62.

Effects of massage on the human lens. (Photographed by Dr. James Wallace.)

author made gentle pressure with a tortoise-shell spoon on the lens-capsule. The capsule remained intact, and, while the partially opaque lens clouded rapidly, there was no inflammatory reaction. Six weeks later the cataract was extracted, and the capsule, after it was gently divided by a cystitome, was left behind in the eye. The figure represents a portion of the lens adjoining the anterior capsule, and shows that such treatment is sufficient to make rents in the softened cortical of incipient cataract, while the amount of granular degeneration along the fissures shows that it could not be due to the cut of the cystitome. The rent here represented runs off obliquely through the lens-fibres, and on each side, more markedly at the upper margin, the fibres are pulled out of place and their frayed and broken ends turned inward, while irregular cavities have formed between them, and the fibres themselves are undergoing a granular degeneration much greater than those in their vicinity which have not been disturbed by the pressure. From this fissure in the lens spaces lead off, running between the fibres, filled (as in many other cataractous lenses) with a granular albuminoid material.

### RATE OF FORMATION OF CATARACT.

In simple senile cataract the rate of progression is very slow, and months and often years elapse between the period of commencement and that of maturity. This is not only true of the form which begins in the peripheral cortical layers, but also of that which commences with perinuclear opacity; indeed, this latter form, which often occurs in myopic eyes, is generally held to be unusually long in ripening. On the other hand, traumatic and complicate cataracts often form with great rapidity. Thus, Ritter[1] reports a case where normal vision was lost and an almost complete cataract was formed in the course of a week, while Hirschberg[2] records the maturation of cataract in twenty-five days. Seely describes a cataract where there was good vision in March, followed by a ripe cataract in May. According to Just,[3] a mother observed a clouding of the lens coming on in her young child during a convulsion, and in another case the eight-weeks-old child of a woman whose two previous children had congenital cataract had its lenses cloud in a few minutes. Two hours later Just saw the child, whose eyes he had previously examined and found to have clear media, and saw that in one of them was a mature cataract and in the other commencing opacity of the lens. Carter[4] reports a case of a girl seventeen years of age in whom cataract developed in fourteen days, and another where in an anæmic and amenorrhœic patient it became complete in three weeks. The rapid maturation of cataract in eyes where it has previously existed is more common. Nettleship relates an instance where marked cataract had existed in the fellow-eye for sixteen years, while in the useful

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[1] Ritter, Klinische Monatsblätter für Augenheilkunde, 1870, viii. 56.
[2] Hirschberg. Nagel, Jahresbericht, 1874, S. 255.
[3] Just, Centralblatt für prakt. Augenheilkunde, Januar, 1880, S. 8–11.
[4] Carter, Lancet, 1881, p. 419.

eye a comparatively clear space in the lower outer portion of the lens allowed the patient to read his letters and the newspapers, but the ability thus to use his eye was lost within three weeks.

### DISEASE OF THE KIDNEY AND ALBUMINURIA AS A CAUSE OF CATARACT. (CATARACTA NEPHRITICA.)

*Diminution of Renal Permeability in Cataract Cases.*—Frenkel has recently experimented on this subject by injecting methyl blue into the muscles of the thigh of cataract patients, and claims that the elimination by the kidneys is slower in them than in persons of equal age not so affected. If elimination was normal in promptness the cataractous patient was found to have diabetes or a cataract due to local eye disease such as iridochorioiditis.

Deutschmann has directed our attention to *the ratio of the occurrence of albuminuria in cases of senile cataract*, and has classified certain cases as nephritic cataract. He found albumin in thirty-three per cent. of his cataract cases. His conclusions, however, as to a special variety of cataract which can be so classified do not seem to be supported by more extended investigations, and there is probably no distinctive nephritic cataract except in so far as extensive and long-continued kidney disease impairs the general nutrition and thus favors the formation of cataract. Ewetsky[1] has added largely to our knowledge of this subject by his careful examinations conducted at the Moscow eye clinic and almshouses. Out of two hundred cases at the eye clinic thirty-eight, or nineteen per cent., had at times small quantities of albumin in their urine, while casts were found in fourteen cases, seven per cent. On the other hand, an examination of ninety cases of renal disease showed retinitis in fifteen per cent. and traces of cataract in 7.7 per cent. In the poorhouse, out of five hundred and eighty-four inmates between fifty and ninety years of age two hundred and sixty-four, forty-five per cent., had traces of cataract. Of these the urine of five hundred and sixty-one cases was examined, and in fifty-nine, 10.5 per cent., albumin was found, while in nine, or 1.6 per cent., casts were present. This would look as if in a large proportion of cases the albumin present was due to prostatitis, cystitis, and other common maladies of old people. Becker[2] in 1888 tells us that of the cataract patients at the Heidelberg clinic six per cent. had albumin in the urine. Some years since I examined over one hundred cases of Bright's disease in general hospitals, the better to study the forms and frequency of albuminuric retinitis, finding twenty-five per cent. affected with it, while in many of the advanced cases of the disease slight opacities of the lens were present, mostly as peripheral striæ.

### DIABETIC CATARACT.

Foerster, in his valuable essay on the relation between eye-diseases and general diseases, has told us that, while in many instances diabetic cataract

[1] Ewetsky, quoted in Nagel, Jahresbericht, 1887, S. 886.

[2] O. Becker, Die Universitaets Augenklinik in Heidelberg, 1888, S. 58.

develops like senile cataract, in the young it often assumes a different type, distinguished by a milkiness immediately under the capsule, with marked differentiation between the sectors of the lens. Becker, however, has shown that in many cases the incipient opacity lies deeper, with a clear layer in front of it, while Horner speaks of the frequent occurrence of punctate and posterior cortical cataract in diabetics. Becker, however, in outlining the appearance of diabetic cataract, says that cloudiness commences in the equatorial zone, spreads thence to the posterior cortical, and afterwards involves the anterior cortical. The opacity usually forms first in the superficial cortical fibres, thus differing from the cloudiness of the cortical which occurs in senile cataract with nuclear sclerosis. This is soon followed by a rapid and considerable swelling of the lens caused by the endosmosis of watery fluid into the capsular sac. The latter author has given us one of the most instructive investigations as to the exact nature of diabetic cataract in the young. The lens was taken from a patient nineteen years old, who died of diabetic coma two days after the performance of a preparatory iridectomy. The epithelium of the anterior capsule is well preserved, and the equatorial lens-fibres are less affected than those between them and the nucleus, where there are numerous more or less spindle-shaped spaces between the fibres, while smaller ones are visible in the nucleus. Between the anterior capsule and the anterior surface of the lens is a large space filled with coagulated albuminous fluid, and a similar one between the posterior surface of the lens and the posterior capsule. Becker sums it up by calling it an œdema of the lens. As to the frequency with which cataract occurs in diabetics, the same author tells us that out of eleven hundred cataract cases operated at Heidelberg, one per cent. had sugar in the urine. Chemical analysis shows that in many instances there is sugar in the lens as well as in the aqueous humor. Deutschmann has objected to this being considered the cause of cataract, because in experiments on animals a much larger percentage seems to be necessary to produce opacity; but the experiments usually spread over only a short time, and there does not appear to be any reason why extremely minute quantities acting over a long period of time should not also produce it. In fact, the cataract of experimental diabetes is always produced rapidly, while clinical observation shows that diabetic cataract usually forms slowly. It is doubtful, therefore, whether the anatomical changes even in the early stages of diabetic cataract are exactly those found in that artificially produced.

Seegen has reported two cases of spontaneous absorption of diabetic cataract, while Tannahill has reported a similar one. Koenig[1] has also observed a clearing up of diabetic cataract where in one eye in the course of two years vision improved from counting fingers to V. = 1/3. In the fellow-eye dense opacity still remained, but it was less than at the previous examination. The same author has also seen a clearing up of cataractous

[1] Koenig, Société Ophthal. de Paris, Annales d'Oculistique, Mai, 1897, p. 398.

opacities in a case of uric acid diathesis, and Despagnet mentioned a resumption of transparency in an arthritic cataract under the influence of Contrexeville waters (observation of Debout d'Estries). Appenzeller records a sharp attack of diabetes lasting fifteen days, when there was an acquired myopia of 1 D. which disappeared with the diabetes, while Risley has reported two cases of sudden transient increase in the refraction of the eye, corresponding with marked increase of sugar in the urine, with subsidence to normal refraction when the sugar diminished.

### ATHEROMA OF THE BLOOD-VESSELS AS A CAUSE OF CATARACT.

While it has long been taken for granted that atheroma and degeneration of the blood-vessels of the eye would, by impaired nutrition of the lens, lead to the formation of cataract, Michel[1] has claimed that atheroma of the carotids stands in close relation to the occurrence of senile cataract, and that one-sided cataract, where the cause is unknown, finds in this condition a satisfactory explanation. Karwat,[2] following up this suggestion, gives twenty cases, in only three of which was there no degeneration of the carotids. Van Brommel[3] maintains similar views, and Nickelsburg[4] states, where atheroma affects the carotid of one side only, that cataract usually develops first in the corresponding eye, while, when it affects equally both carotids, both eyes are simultaneously affected. Weil,[5] however, after careful examination of fifty-three individuals with cataract, found only sixteen affected with atheroma of the carotids, while in thirty-seven no such affection could be demonstrated. In six of the sixteen cases in which atheroma was demonstrated, the disease of the artery was on the same side as the first-developed cataract, while in the other ten the reverse was the case. Weil calls attention to the impossibility, even in emaciated people, of determining the state of the internal carotid by palpation, and to the difficulty of doing so in the common and external carotid. Knies also maintains the contrary view, and has often by careful palpation demonstrated that the greater development of the atheroma of the carotids was on the side on which cataract had not developed. My own experience would indicate that atheromatous arteries are quite common in patients with cataract, but that while atheroma of the radials is frequent in patients with cataract, and a similar state of the temporal artery not uncommon, atheroma of the carotid of sufficient extent to be detected by palpation is rare, and it seems doubtful whether its presence is

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1 Michel, Das Verhalten des Auges bei Stoerungen im Circulations gebiete der Carotis, Festschrift zu Ehren Prof. Horner, 1881.

2 Karwat, Beiträge zur Erkrankung des Auges bei Carotis Atherom, Inaug. Diss., Würzburg, 1883.

3 Van Brommel, Zur Etiologie der Cat. Senilis, Inaug. Diss., Würzburg, 1889.

4 Nickelsburg, Weitere Beiträge zur Etiologie der Cataracta Senilis, Inaug. Diss., Würzburg, 1892.

5 Weil, quoted by Becker, Anat. der gesunden und kranken Linse, 1883, S. 128.

of any value in the prognosis of cataract except as a possible indication of diseased conditions of the walls of the intra-ocular vessels.

### FREQUENCY OF CATARACT.

The more one examines the eyes of elderly people the more one is struck with the extreme frequency with which some degree of opacity of the lens is found,—so much so that one is almost inclined to agree with the famous dictum of Walther, "that every one becomes cataractous who does not die prematurely." On the other hand, in only a very small percentage of these cases does the cataract ever advance far enough to prevent useful employment of the eyes. Of those who have the lens opacity sufficiently developed to cause their complaint to be entered in the books of an eye hospital as cases of incipient cataract, we find in various clinics a percentage varying from 5 per cent. to 10 per cent. Thus, Dor[1] out of 8008 cases gives $8\frac{4}{5}$ per cent. of lenticular opacity. Knapp, out of 10,004 cases in Europe, gives 7.6 per cent., while in New York, out of 6379 cases, he finds but 5.5 per cent. Wecker[2] gives 12 per cent. as the number occurring at his clinic. Rydel,[3] in Cracow, reports 7.65 as the proportion, while Cohn's statistics give 6 per cent. Schoen[4] out of 6689 eyes of all ages examined for lenticular opacity gives 18.4 per cent. as having greater or less degrees of cataract. Of these cases a very small proportion are mature cataract. Thus, if the number of extractions represents, as it nearly does, the number of ripe cataracts at the Wills Hospital, we have in twenty years, from 1872 to 1891 inclusive, 129,806 cases of eye-disease and 1428 extractions, making 1.1 per cent. of mature cataract; while if the later years, where the number of patients is much larger, are taken, we have a still smaller percentage. Thus, in the five years from 1886 to 1891 inclusive, we have 53,453 cases of diseases of the eye and 488 extractions, a percentage of 0.9. To make this absolutely fair the congenital and infantile cataracts which were discinded should be added, and in the twenty years there were 205 of these, giving the percentage of congenital and infantile cataract as 0.01.

### THE AGE AT WHICH UNCOMPLICATED CATARACT IS MOST COMMON.

While, as previously stated, cataract often comes on without *demonstrable* cause, either local or general, nevertheless it is certainly influenced by the general health, coming on earlier when the nutrition is impaired, especially in those cases where the lowered vitality has produced local changes and degenerations in the blood-vessels of the uveal tract. According to Arlt, out of 882 cataract patients between the ages of twenty-six and eighty-two, 626 were between forty-five and seventy years of age. It is, I think, the general experience that it is more frequent and develops earlier in farm-

[1] Dor, Nagel, Jahresbericht, 1837, S. 319.

[2] Wecker and Landolt, Traité Complet d'Ophtalmologie, vol. i. p. 847.

[3] Rydel, Klinische Monatsblätter für Augenheilkunde, 1879, S. 90–94.

[4] Schoen, Funktions Krankheiten des Auges, 1893, S. 158.

ers and day laborers than it does in those classes of society who are less exposed to exhausting physical labor and to intense glare and heat. According to Hirschberg, there are many more cataracts ripe in the farming class at fifty years than are to be found in city dwellers at the same age. He also tells us that climate with exposure to light and heat have much to do with it, either from direct action on the eye or by the state of nutrition induced by high heat, severe manual labor, and scanty nutriment, and that in the East Indies the majority of cataract patients come to operation at forty years or thereabouts, while in Europe the majority of extractions are at sixty-two years of age. Jackson found that out of 1545 patients at the Wills Eye Hospital at Philadelphia over fifty years of age, 449 had some lens opacity. Arranged in five-year periods the percentages showing such opacities were as follows: 15 per cent. between fifty and fifty-five, 16.1 per cent. between fifty-five and sixty, 30.2 per cent. between sixty and sixty-five, while in the ten-year period between sixty-five and seventy-five he found 77 per cent.

## STRIATED KERATITIS AFTER CATARACT OPERATIONS.

This form of keratitis is of not infrequent occurrence and has long been familiar to clinicians. It is not, however, peculiar to cataract operations, but is found also in inflammatory affections of the cornea. Raehlmann [1] gives an excellent description of it, both as occurring after linear operation for cataract and in rodent ulcer. When present, it develops after closure of the wound, and consists of grayish stripes situated in the deeper layers of the cornea and running at right angles to the direction of the wound. At times these are joined by cross-lines, making an irregular net-work. There have been but few pathological examinations of such opacities recorded. Becker [2] found in one case swelling of the deeper layers with a massing of lymphoid cells in the enlarged interstices of the tissue. Laqueur and Recklinghausen [3] found a hyaline degeneration of the protoplasmic elements with secondary distention of the lymph-passages. According to Mellinger,[4] the use of cocaine causes swelling of the lips of the corneal wound with some shedding of the epithelium, but never a striated keratitis, while sublimate solutions constantly cause striped keratitis, due to cloudy swelling of the corneal tissue, more especially of the corpuscles and their protoplasmic branchings, which become thicker and more wavy than in normal tissue. While it must be considered demonstrated that the corneal tissue changes in striped keratitis, which have been described by Becker, Recklinghausen, and others, really exist, nevertheless it is difficult, as was pointed out by Laqueur, to understand how such minute changes could give rise to the clinical appearances as we find them after cataract operations and in some

[1] Raehlmann, Klinische Monatsblätter, 1877, S. 1–21.

[2] Becker, Atlas der pathologischen Topographie des Auges, 1887, Bd. iii. S. 83.

[3] Laqueur and Recklinghausen, Bericht der Heidelberger Congress, 1887, S. 116.

[4] Mellinger, Archiv für Ophthalmologie, xxxvii, 4, S. 159.

corneal ulcers. Nuel[1] was the first to prove that the microscopic appearances are due to the production of ridges in the membrane of Descemet and in the immediate underlying layers of corneal tissue. He found that this variety of corneal opacity is due to a folding of the cornea (which he attributes to too tight bandaging), which produces cracks in the posterior corneal epithelium (membrane of Descemet) and a subsequent œdema of the corneal tissue. The diffuse opacities of the cornea which come on in from two to five days after operation he attributes to the use of solutions of sublimate, which, according to his view, do not need to be injected into the anterior chamber to produce them. More recently Hess[2] has given additional evidence in favor of this view by a careful examination of a human eye which had been operated on for cataract, as well as by a study of experimental striated keratitis in rabbits. Schirmer has since published a paper on the subject, in which he pictures the folds of the membrane of Descemet and the immediately underlying corneal layers as occurring in experimental cataract operations on rabbits and in artificially produced hypopyon keratitis, and also a similar state of affairs which he found in a wounded human eyeball which went on to suppuration in a case of hypopyon keratitis. He further mentions appearances in the corneæ of shrinking eyeballs which were clinically similar to the usual appearances of striated keratitis, but which lay near the anterior surface of the cornea, and were due to somewhat similar foldings of the membrane of Bowman. Fig. 63 (after Hess) shows a magnified view of the foldings of Descemet's membrane, while the other (Fig. 64) drawing on the same plate represents the situation of the incision in the cornea and a series of folds extending from it, as seen under a lower magnifying power.

Fig. 63.

Folds of Descemet's membrane in striated keratitis. (Hess.)

Fig. 64.

Folds of Descemet's membrane in striated keratitis. (Hess.)

[1] Nuel, Congrès de la Société Française d'Ophth., May, 1892.
[2] Hess, Archiv für Ophthalmologie, 1892, xxxviii. S. 4.

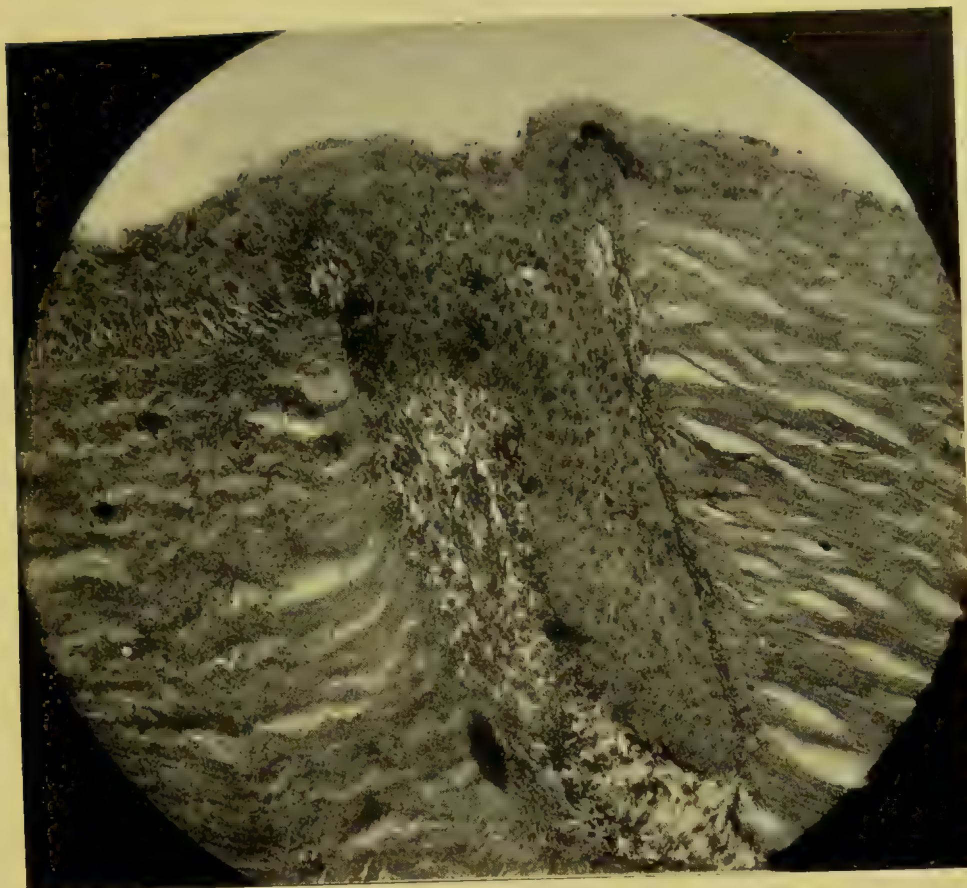
## ON THE NORMAL HEALING OF WOUNDS MADE IN CATARACT OPERATIONS.

In normal cataract operations where the incision is in the corneal tissue the lips of the wound lie more or less accurately in apposition, and there occurs a felting of the swollen ends of the corneal fibres of one lip with those of the other. This is usually most marked in the central layers of the cornea, the anterior and posterior layers retracting to a greater extent. This felting of the corneal fibres is assisted by a fibrinous exudation, and constitutes the first stage of repair, being usually sufficiently complete in from twenty-four to forty-eight hours to retain the aqueous humor and thus permit its accumulation and the re-establishment of the anterior chamber. Within a few hours the anterior epithelium commences to proliferate and grows down into the wedge-shaped space between the lips of the wound, and by the fourth day has usually filled out this space and united with the epithelium on the other edge of the wound. By this time a plug of fibrin has also formed on the posterior edge of the wound, and the union has thus become fairly firm, although still readily burst open by any undue pressure of the lids, by coughing, or by any other violence. Later fibrous tissue and blood-vessels, shooting in from the periphery, form in the wound, and some time between the second and the sixth week cause a firm and permanent union. Even at this latter stage, however, it has by no means attained its eventual consistence and strength, and I have known a severe accidental blow at this period to burst open the wound and evacuate a large portion of the contents of the eyeball. As is well known by every clinician, the period of repair varies greatly in different cases. Figs. 65 and 66 give a photographic representation of a section through the extraction wound nine weeks after the operation. They show respectively the upper and lower halves of the incision. In the upper half there is a double epithelial plug extending inward from the corneal epithelium, that in the peripheral lip of the wound reaching much farther down into the wound. This plug is in the processs of absorption, and is being replaced by fibrous tissue. In the lower half of the wound the frayed and bent edges of the corneal lamellæ are united to those on the other side of the incision by a newly-formed mass of fibrous tissue. The wound in the membrane of Descemet is covered by a fibrous cap which at its centre is adherent to the hyaloid membrane of the vitreous. It was long believed that the cut edges of the membrane of Descemet never reunited, and they probably never do so directly, but Gepner[1] and Wagenmann[2] have lately shown that the endothelial cells of the membrane proliferate, and form a thin vitreous membrane which often in time glues together securely the cut edges. Where the wound is in the sclerotic and there is no conjunctival flap the process of healing is very much like that of a corneal wound, but where a considerable

[1] Gepner, Archiv für Ophthalmologie, 1890, xxxvi., 2, S. 255.

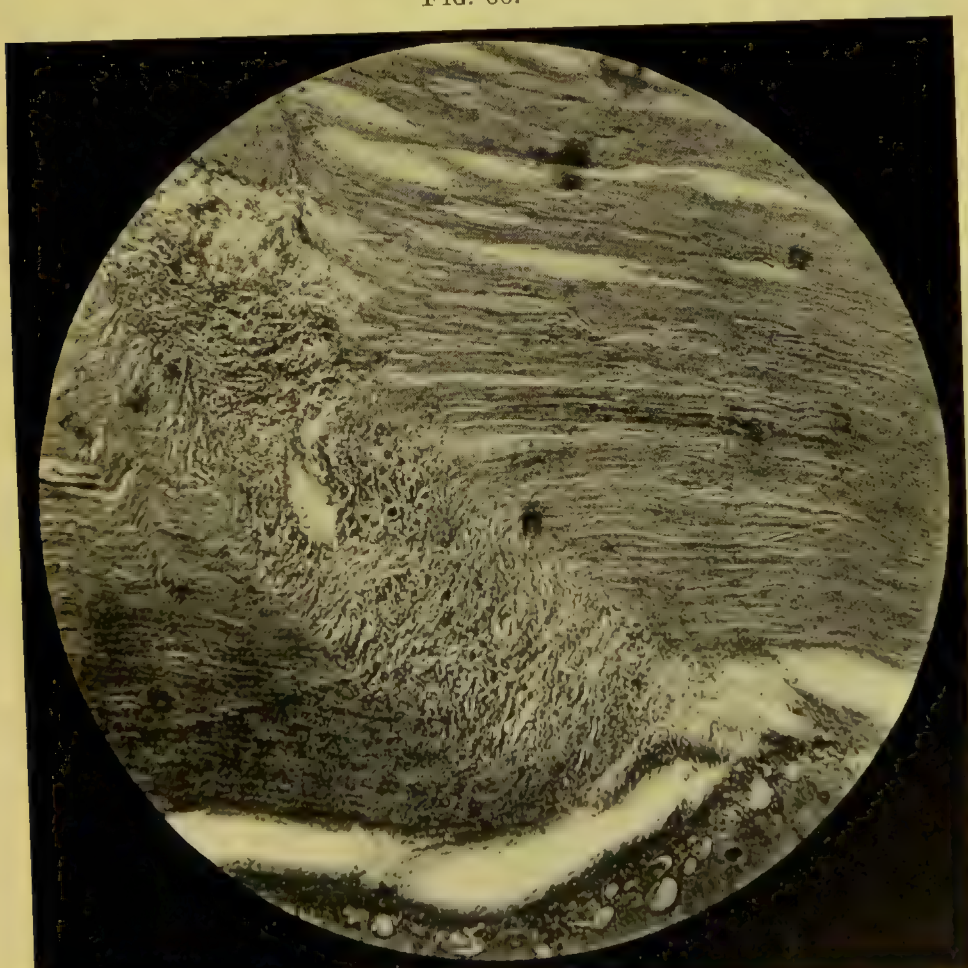
[2] Wagenmann, ibidem, 1891, xxxvii., 2, S. 21.

Fig. 65.



Upper half of extraction cicatrix nine weeks old. (Photographed by Dr. George W. Norris.)

Fig. 66.

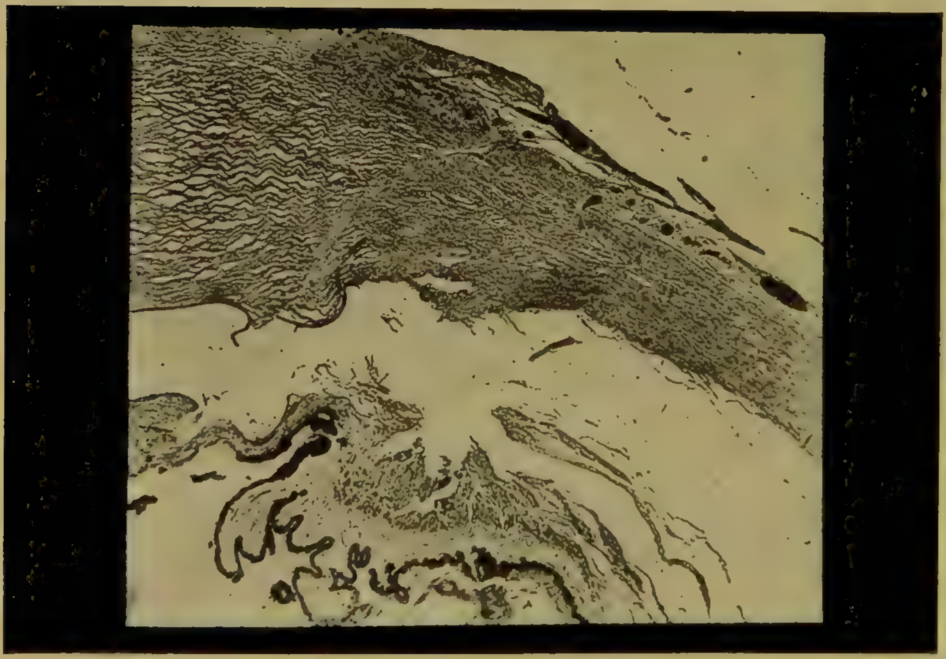


Lower half of extraction cicatrix nine weeks old. (Photographed by Dr. George W. Norris.)

FIG. 68.

Displacement of lips of the wound, with cystoid cicatrix.

FIG. 69.



Cicatrix after sclerotomy.

flap exists an epithelial plug forms only at the point where the divided conjunctiva unites, and the closure of the wound is mainly due to effusion of lymph and felting of the ends of the cut scleral fibres. Where there is any considerable gaping of scleral wounds Lubinsky[1] has shown that the healing is mainly due to the subconjunctival and chorioidal tissues, which form an intercalated plug. Where any foreign substance comes to lie between the edges of a cataract incision, such as remnants of lens-matter, a portion of the iris, a tongue of capsule, or a loop of vitreous, the firm and permanent healing is always delayed, and on any pressure on the eyeball or rise of intra-ocular tension there is a slight leakage of aqueous, separating the superficial fibres, and often laying the foundation either for a filtration cicatrix or for a staphylomatous projection.

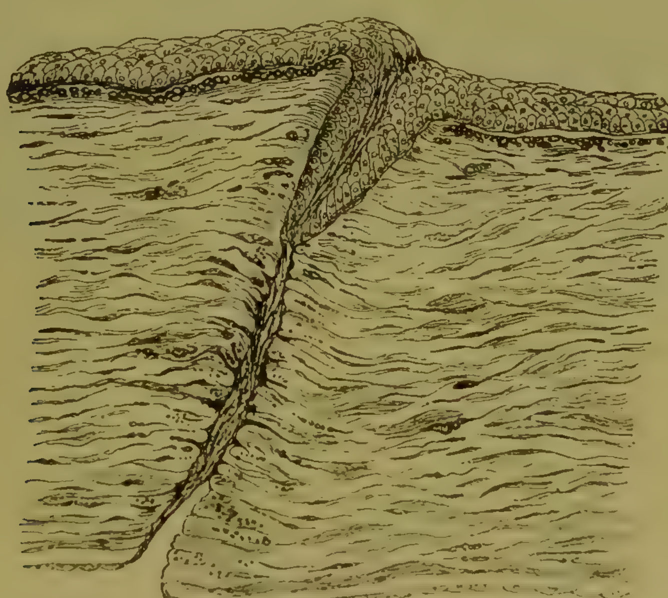
### RELATIVE POSITIONS OF THE LIPS OF THE WOUND IN THE CICATRIX.

This varies very much in different cases even where the healing is clinically a normal one. In some few instances the lips of the wound after complete cicatrization are almost in their original position, separated only by a thin layer of cicatricial tissue, but in most instances the distal lip over-rides the proximal to a greater or less extent, and this with pull of subsequent cicatrization produces a flattening of the cornea in a meridian at right angles to the incision. The intra-ocular pressure has also a tendency to cause an increase in the curvature of the horizontal meridian.

### DISPLACEMENT OF LIPS OF THE WOUND AFTER CATARACT EXTRACTION.

Fig. 57, at page 311, from Becker, shows a wound in the cornea from

FIG. 67.



Cicatrix after cataract operation; riding up of peripheral lip of the wound. (Becker.)

flap extraction which has healed in a nearly normal position, while Fig. 67, from the same author, shows an upriding of the lower lip of the

[1] Lubinsky, Archiv für Ophthalmologie, 1867, xiii., 2, S. 377.

wound. Fig. 68, taken from one of my cases of glaucoma, exhibits a slight displacement of the flap and a projection of the proximal end into the anterior chamber. It is also a good example of cystoid cicatrix. The iris has prolapsed into the wound, and has prevented almost entirely any union of the corneal tissue, the closure having been effected mainly by the epithelial plug, with slight amount of fibrous tissue uniting the sub-epithelial layers of the cornea. In contrast with these methods of healing, Fig. 69 shows the dense cicatrix of a sclerotomy in the corneo-scleral junction, but at the same time its great effect in loosening up the adjacent fibres of scleral tissue. Fig. 70, which gives a less magnified view of the section previously shown in Figs. 65 and 66, affords an entire view of the line of incision, and shows well the characteristic displacement of the distal lip of the wound.

## ZONULAR CATARACT.

Since Jaeger, in 1868, first demonstrated the true nature of zonular cataract it has been a favorite subject of study by ophthalmologists, and large numbers of cases have been reported, with a careful description of the appearances as seen by the use of the ophthalmoscope and oblique light. It is, however, only within the last few years that, by the careful study of thin sections of such lenses, our knowledge of the pathological changes has advanced beyond the statements given us by Jaeger. To Deutschmann, Beselin, Lawford, Schirmer, Hess, Peters, and E. Treacher Collins we are indebted for careful microscopic examinations of this form of cataract. Deutschmann[1] found a double layer of opacity enveloping an intact nucleus, and covered in turn by clear cortical, the clouding being apparently due to finely granular detritus collected in fissures between the fibres. Beselin[2] and Lawford[3] obtained similar results, except that they both observed more or less degeneration of the nucleus. Schirmer,[4] who had an opportunity of investigating six cases, thought that the main cause of the opacity was innumerable small dots and vacuoles which existed everywhere around the periphery of the nucleus. Both the nucleus and the cortical contained also opacities of less extent and not sufficient to make them appear cloudy either with oblique light or the ophthalmoscope. There was but little separation of the layers of the lens-fibres from each other. In one of these cases there was also well-marked anterior capsular cataract. Hess[5] considers the true perinuclear opacity as caused by small, irregularly round vacuoles, whose equatorial diameter varied from 0.002 to 0.01 millimetre, and thinks that the angular or rafter-like opacities at the equator of the nucleus are caused also by vacuoles. In a case where

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[1] Deutschmann, Archiv für Ophthalmologie, xxxii., 2, S. 295.

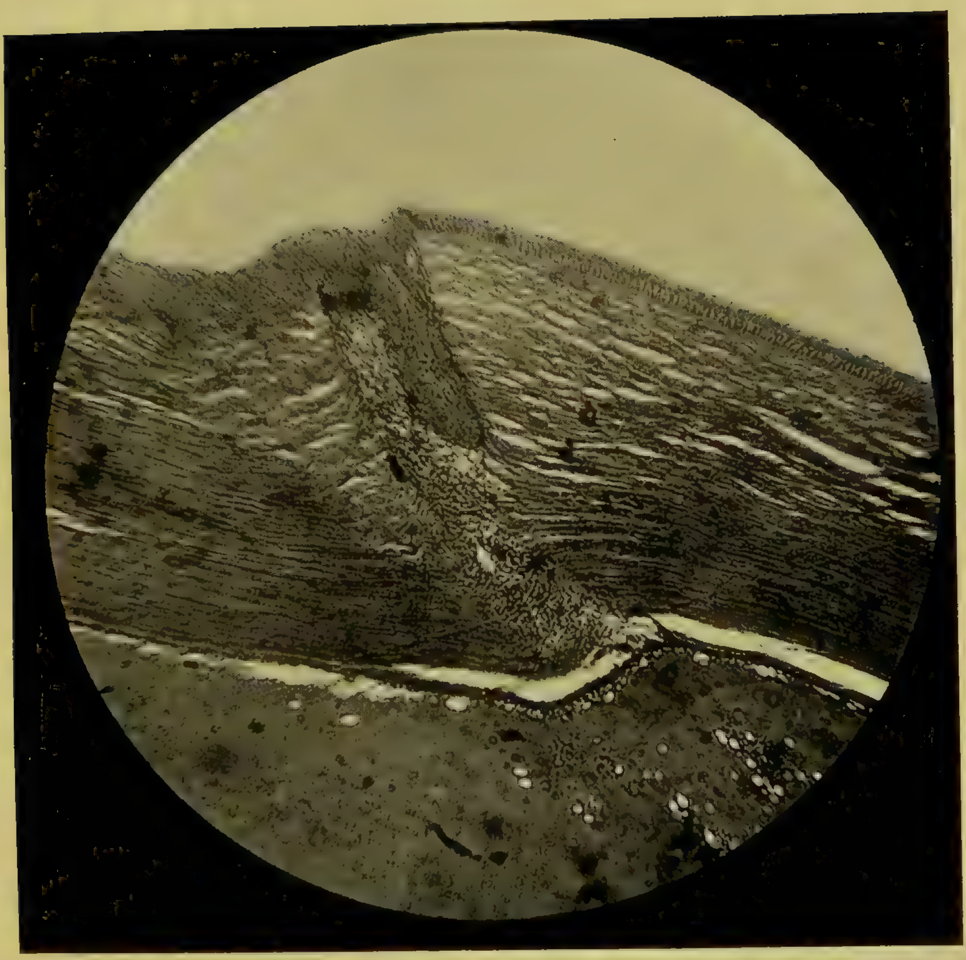
[2] Beselin, Archiv f. Augenheilkunde, xviii., S. 71–86.

[3] Lawford, R. L. O. H. Rep., vol. xii., 2, p. 184.

[4] Schirmer, Archiv für Ophthalmologie, xxxv., 3, S. 147.

[5] Hess, ibidem. xxxix., 1, S. 183.

FIG. 70.



Cicatrix nine weeks old. (Extraction April 10, 1895, enucleation June 5.)
(Photographed by Dr. George W. Norris.)

Fig. 71.

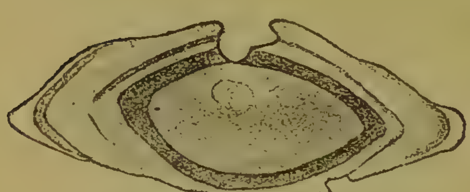
Section of zonular cataract. (Photographed by Dr. James Wallace.)

punctate cataract was coexistent with zonular, he found club-shaped and irregularly-ovoid opacities between the lens-fibres, and to them he attributes the punctate dots. The arrangement of the minute dots in this instance corresponded with the distribution of the lens-fibres, and not with the intersectorial spaces. Peters gives drawings of a case where in one eye the central part of the nucleus has fallen out in the preparation, and where in both there is enormous separation of the nucleus from the cortical layers by large, irregularly-shaped cavities filled with fluid. According to Collins, who has examined microscopically seven zonular cataracts, the pathological changes are of three sorts: first, fissures between the lens-fibres which run concentrically with the nucleus, and which may or may not be filled with granular material; second, small vacuoles, of an average size of 0.005 millimetre, filled with hyaline material; third, larger spaces, averaging 0.02 millimetre in diameter, filled with granular material. These, he considers, correspond to the radiating spokes. In one of the seven cases the nucleus was entirely clear. In the remaining six there were vacuoles in it. In two these were distributed pretty evenly throughout the nucleus.

Fig. 71 was taken from a case in my own practice. The lens was extracted from a man, aged thirty-five, with zonular cataract, in whose family cataract was hereditary, and whose twelve-year-old niece was at the same time under my care for zonular cataracts on which I had performed discissions. The plate is a reproduction of a photomicrograph, and shows that the nucleus of the lens exhibits small, irregularly-rounded dots of degeneration, some of these dots being filled with granular material, while others have bright, highly-refracting contents; that minute spots, filled with granular material, are the cause of the perinuclear opacity; that the cortical fibres adjacent to the nucleus are fairly clear, although they exhibit slight, irregularly-rounded spots of degeneration, and that the whole mass is moderately coherent, with but little formation of cracks or cavities between the lens-fibres, those near the posterior surface being evidently artificial and due to the drag of the cutting microtome. Between the peripheral fibres may also be seen in many places elongated masses of granular material. These are sufficiently numerous to form an outer, although incomplete, zone of opacity. The nucleus itself is irregular in shape, and, instead of being ovoid, is irregularly prominent at its posterior surface, while the adjoining lens-fibres show a similar angular curve. At the summit of this curve, as well as at the equator of the nucleus, both above and below, there is a mass of granular opaque material. The layers of lens-fibres immediately surrounding the nucleus are comparatively healthy, while both in the anterior and posterior cortical they are more granular, and are separated in many places by granular albuminous material. A somewhat higher magnification shows in the nucleus of the lens evidences of a similar but less marked degeneration, — viz., minute, irregularly-rounded areas of disintegration, some of which are

filled with granular material, while others have contents of a high refracting power. Figs. 72 and 73 show, slightly more magnified, the appearances of the opaque band of a zonular cataract extracted from another patient. There is much less granular material in it than in the first case, and many more drops of a highly-refracting clear material of various sizes. We thus have the opportunity of examining what is probably the same process in different stages. In the earlier stage the material in the affected zone, viewed with a low power, looks granular, but, examined with an immersion-lens, it proves to be composed of round masses, of which the material is cloudy and finely granular, while towards the outer edge and scattered through it are nucleated cells in process of degeneration, possibly remnants of imperfectly-formed epithelial cells thrown off by the capsular epithelium at the time at which it was in contact with the diseased zone and before the comparatively healthy external layer had formed. In the second specimen we have a more advanced stage of the process. No distinct cells with cell nucleus are to be seen, but we have rounded spaces filled with some highly refractive oily material. The fact that there may be in the same lens a double, or even a triple zone of opacity, separated each from the other and from the nucleus and anterior capsule by a layer of clear or almost clear lens-matter, is within the experience of almost every practitioner, and has been repeatedly demonstrated clinically and anatomically.

FIG. 74.



Triple zonular cataract. (Schirmer.)

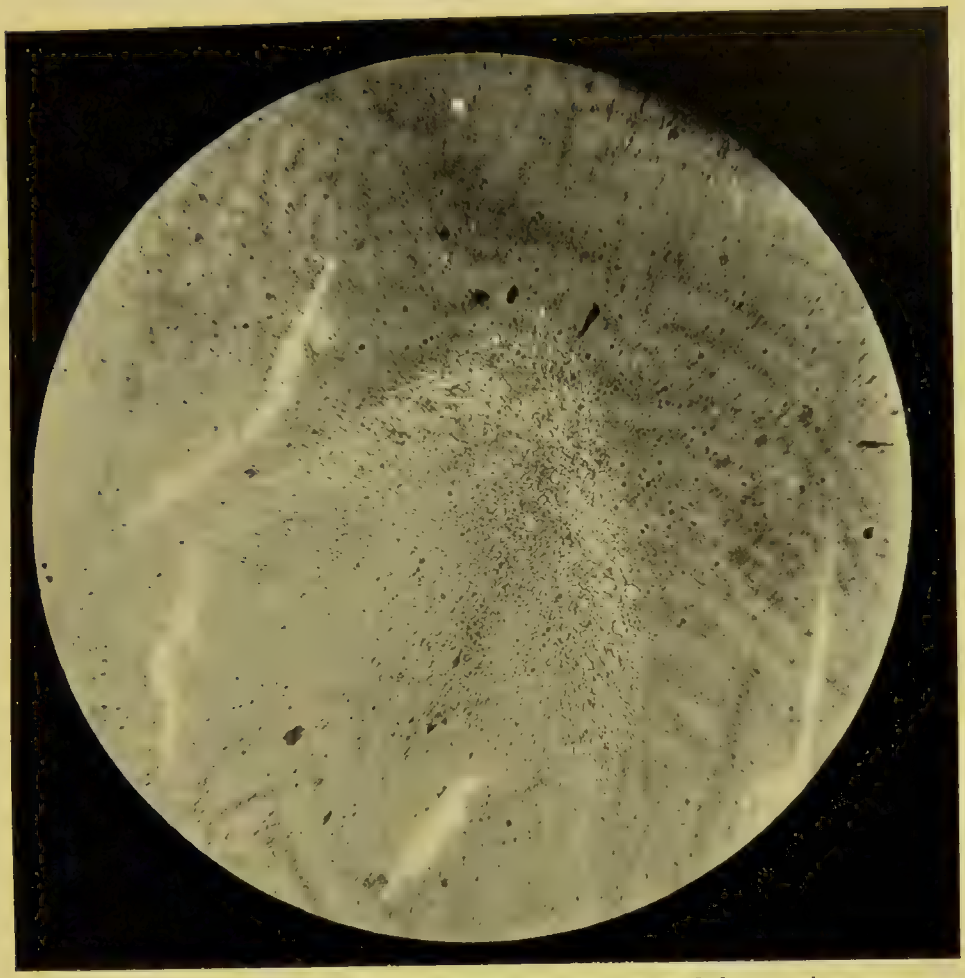
Fig. 74 is a reproduction of a section of triple zonular cataract given by Schirmer.[1] In cases where there is a single zone, Dub,[2] who has measured ten cases in the living subject by an ingenious application of the method of double view, gives the equatorial diameter of the opacity as varying from 4.4 to 5.6 millimetres. Direct measurements have been made, either after extraction of the lens or post mortem, by several authors, whose results are herewith tabulated.

| | Millimetres. |
|---|---|
| Beselin gives the diameter of the opaque zone as | 6 |
| Lawford, one case | 5.25 |
| Lawford, two cases | 4.5 |
| Schirmer, one case | 5.75 |
| Schirmer, one case | 6 |
| Schirmer, one case | 3.25 |
| Collins, two cases | 3.5 |
| Collins, one case | 5.5 |

Where there is a second or a third ring the diameter has at times been found to be as much as eight millimetres, while according to Dub the equa-

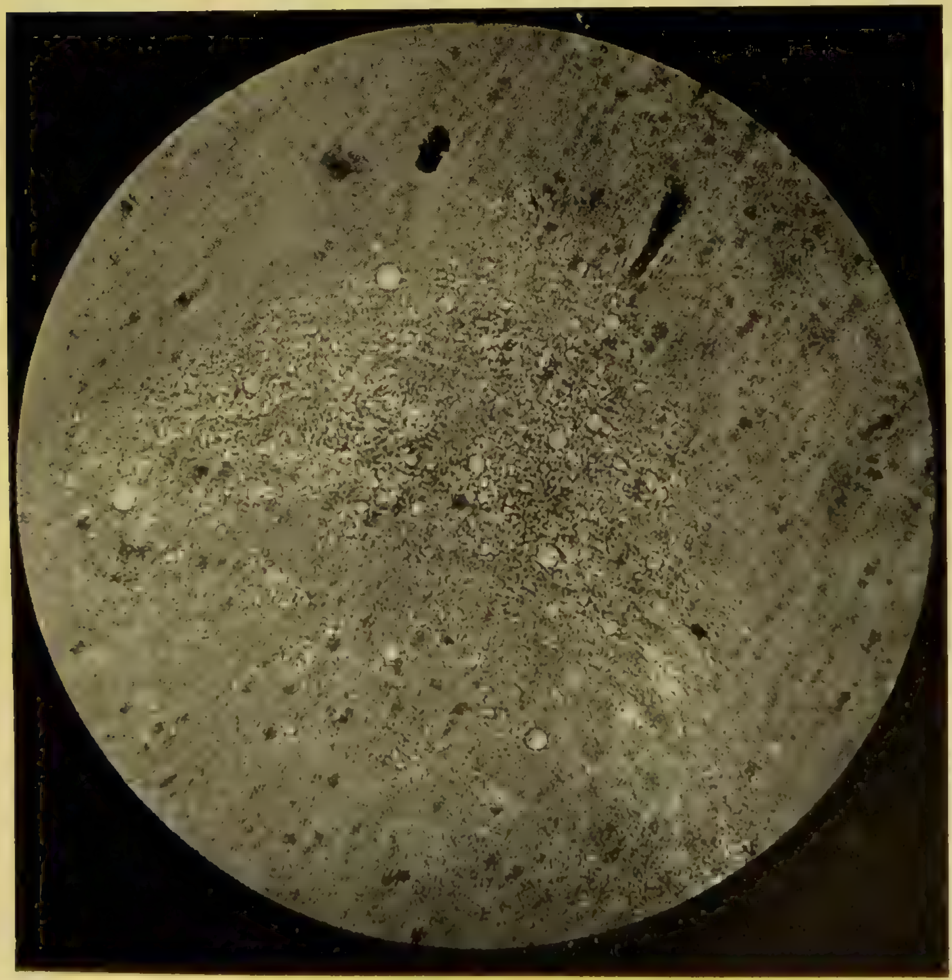
[1] Schirmer, Archiv für Ophthalmologie, xxxv. 3.
[2] Dub, ibidem, xxxvii., 4, S. 26.

Fig. 72.



Section of a zonular cataract showing equator of the zonule.
(Photographed by Dr. George W. Norris.)

Fig. 73.



Section of a zonular cataract showing equator of the zonule.—More highly magnified.
(Photographed by Dr. George W. Norris.)

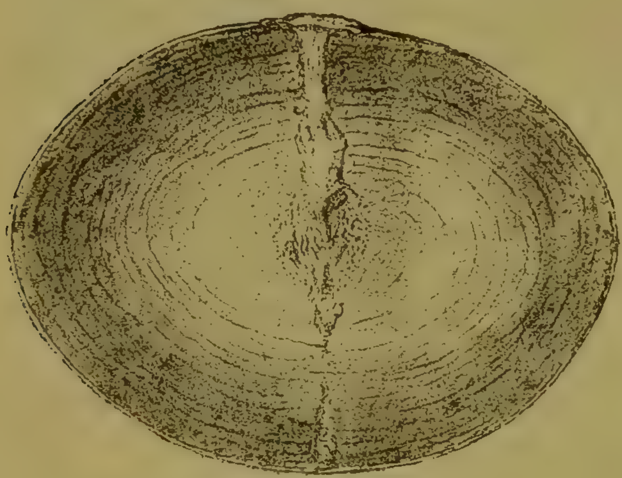
torial diameter of the lens of a child of ten to eleven months is 7.46 millimetres, which Jaeger gives as the measurement of the lens of the newly-born. If, as seems fair to assume, we suppose that some disturbance of lenticular nutrition occurs at an early stage of fœtal life while the direction of the lens-fibres is axial; that we may have cloudiness of them and some form of axial cataract; that when concentric fibres are added to the lens a like disturbance might give us a nuclear cataract, while a little later after a clear nucleus had once been formed similar nutritive disturbance would give us the usual one-zoned band of opacity; similar disturbances of nutrition occurring even after birth in the superficial layers of the lens where the epithelium is constantly producing new fibres would produce the larger zones of seven to eight millimetres, and there might still be vital force enough in the epithelium under better conditions of nutrition to wrap them in a layer of healthy and transparent fibres. Foerster has long ago called our attention to the similarity of the process of the formation of perinuclear cataract to that of zonular, and I myself had an opportunity of seeing a finely granular double zone of opacity form in the lens of a patient sixty years of age, where the accompanying swelling of the lens produced considerable myopia, as is detailed in a subsequent section under the head of Second Sight. The only essential difference between the formation of such layers in fœtal life and early childhood and those occurring later seems to be that in the one case the epithelium possesses enough vital force to form, under improved nutrition, an overlying layer of healthy fibres, while in the other the germ force is not sufficient to do so, and we soon have the formation of other varieties of opacity in other parts of the lens. The theory that convulsions would cause such violent cramp of the ciliary and lid muscles as to produce a sort of massage of the soft, newly-formed lens-fibres against the comparatively hard nucleus, as held by Arlt, Horner, Schoen, and others, looks plausible, and the intermittence of the convulsions might on this theory be held to correspond to multiple layers of opacity in the lens; but while the frequent occurrence of convulsions in rhachitic children is everywhere admitted, their considerable number ought to produce either more frequent layers of opacity or total cataract. According to v. Arx, out of one hundred and eighty-nine cases of zonular cataract, one hundred and seven, or 56.61 per cent., had been subject to convulsions, and 5.9 per cent. had zonular cataract in both eyes. Certain it is that the most careful inquiry into the history of patients in the large proportion of cases fails to elicit any evidence of previous convulsions or spasms, and a more extended examination shows that a very large proportion of the cases of zonular cataract are certainly congenital.

## AXIAL CATARACT.

Axial cataract may be either congenital or acquired by pathological processes started by ulceration of the cornea or injury to the capsule (Knies, Leber). As was mentioned in discussing the formation of zonular cataract,

it is probable that a disturbance of the nutrition of the lens in early fœtal life, while the lens-fibres run in an axial direction, may cause it. When there is a combination of anterior polar and zonular cataract Knies and Leber think that the newly-formed transparent fibres are hindered from joining by the adhesion in the axial line between the two cataractous zones, and consequently the anterior polar opacities will gradually diminish in size, and there will be only a thread running back to the zonular opacity. Hess, owing to examination of the chicken embryo, is of the opinion that axial cataract is due to delayed union of the mouth of the lenticular sac, and that all the then formed fibres become more or less opaque, especially the strands running from the nucleus to the point where the sac has finally closed. Bach reports an axial cataract occurring in the lens of a medium-sized and apparently healthy rabbit. The opaque band ran from an anterior capsular cataract to a thicker part in the nucleus, and was thence continued by a thread to the posterior pole of the lens, where it again broadened out. The opaque band consisted of degenerated lens-fibres with fine black pigment in places, and at the sides quantities of small vacuoles and myelin masses. Fig. 75 shows a nuclear cataract of a rabbit with opaque bands running to the anterior and posterior poles (after Bach).

FIG. 75.



Axial cataract. (Bach.)

## THE VARIOUS FORMS OF CATARACT CONSIDERED FROM A CLINICAL STAND-POINT.

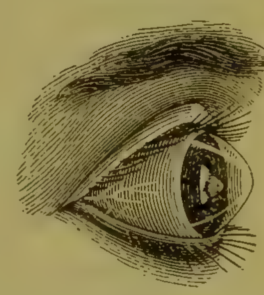
I. **Congenital Cataract and the Cataract of Infancy.**—There is naturally some difficulty in drawing the line between congenital cataracts and those forming during adolescence, as it is comparatively rarely that newly-born children are examined with the ophthalmoscope, and except where the cataract is so marked as to attract immediate attention the eyes are not examined until it becomes evident during adolescence that the acuity of vision is less than normal, and at this period it is difficult to prove that the opacity of the lens has not developed after birth. Enough cases have, however, been observed, and the observation in some instances substantiated by autopsies, to prove that the varieties of lens opacities enumerated under this head are usually prenatal in their incipiency, and due to disease of the fœtus or to its faulty development.

II. **Axial Cataract.**—In this form we have an opacity extending in the direction of the antero-posterior axis of the lens which may go completely

through it or may occupy only a portion of the axis. A cataract of this variety is often irregularly spindle-shaped, and may exist alone or be accompanied by opacities in other parts of the lens. If the opacity be dense and the rest of the lens clear there may be tolerable vision, although Knies[1] has shown how much it interferes with the change of shape of the lens under the influence of the ciliary muscle, and diminishes the range of accommodation. A somewhat similar form of opacity may at times be developed after birth, and it is then usually accompanied by central capsular cataract. Axial cataracts probably date from that early period of development of the lens at which, the lens vesicle having closed, long straight cells push forward from the posterior capsule to meet the growing cylindrical epithelium from the inner surface of the anterior capsule, or, as Hess suggests, and as has been stated with more detail in the section on pathology, may be due to abnormal delay in the closure of the lens sac. In cases undoubtedly congenital it has been found associated with zonular, with anterior polar, with posterior polar, and with central cataract.

III. **Congenital Anterior and Posterior Polar Cataract.**—*Congenital anterior polar cataract* is probably of two varieties, one due to an adherence between the pupillary membrane and capsule, the other due to fœtal inflammation, a plug of lymph having been deposited on the latter. Figs. 39 and 40, at page 301, give a good example of the former variety, while Fig. 76 (after Wilde) is a characteristic picture of the latter. In either case the tissue adherent to the outer surface of the anterior capsule goes on to become atrophic and thinner, but it interferes with the nutrition of the capsular epithelium so as to cause a proliferation of its cells, and a cone of opaque tissue develops, projecting from the inner surface of the capsule into the lens-substance. In the latter method of production we have in fact the same process which we are able to trace in later life as causing acquired anterior central capsular cataract.

FIG. 76.



Congenital anterior polar cataract in a case of conical cornea. (Wilde.)

The *congenital posterior polar cataract* is small, white, and glittering, and we can at times see with the mirror that a thin conical process runs out backward from the lens into the vitreous. In Arlt's clinic, in attempting to discind such a cataract, the little, hard mass was pushed into the vitreous, and then for the first time the retained hyaloid artery could be seen running to it. These opacities have been proved by Berthold[2] to be due to disease and retention of the hyaloid artery and its branches on the posterior capsule. He found that the tissue of the little pyramidal mass was finely striated, with blood-vessels ramifying in it. When it was detached from its position it carried with it a piece of the posterior capsule

[1] Knies, Archiv für Ophthalmologie, xxiii., 1, S. 211.
[2] Berthold, ibidem, xvii. S. 174.

and left a concavity in the posterior layer of the lens. Von Ammon [1] has demonstrated a similar state of affairs in the rabbit, and H. Müller [2] in the goat. This form of cataract is very distinct from the stellate form, occurring also at the posterior pole where the opacity is seated in the layers of the posterior cortical. These latter forms are at times observed at birth and in early life, and are symptoms of preceding retinal or chorioidal disease. In later life they are apt to occur in cases of typical pigment degeneration of the retina and in many other varieties of chorio-retinal disease.

IV. **Zonular Cataract.**—By far the most frequent form of partial congenital cataract is that known as zonular cataract, where a clear or partially cloudy nucleus is enveloped in a layer of opaque and cataractous lens-fibres, which are again covered by a clear and apparently normal cortical. Sometimes two or three such opaque zones can be demonstrated, each separated from the underlying one by a layer of transparent fibres. In such cases, if the opacity is slight and thin and the superficial clear layers of the lens have sufficient depth, the pupil looks normal, and it is only when the patient complains of dim vision, and we examine the eye with the ophthalmoscope or oblique light, that the cataract is discovered. Von Jaeger [3] was the first to give a correct description of this variety of cataract, and to confirm it by an examination of the extracted lens. He describes it as a uniform gray opacity of a single layer of lens-tissue enveloping a clear nucleus and covered by a layer of clear cortical. He had observed it "seven times in four individuals." The equatorial diameter of the hazy zone varied in the different cases from three to five millimetres. A year later Graefe tells us that this is the most common form of cataract in childhood. Arlt had previously described it as stationary nuclear cataract, not realizing the clear centre. The equatorial edge of the opacity may be either smooth and round, or may send out opaque striæ towards the equator of the lens. With the ophthalmoscope we may differentiate it from nuclear cataract by the fact that the opacity seems clearer in the centre and denser in the periphery, where the opaque layers of the anterior and posterior parts of the lens approach each other, while in nuclear cataract the opacity is of course denser at its centre. Zonular cataract may occur without other lenticular opacities, but is at times accompanied by greater or less development of axial cataract. The peripheral edge of the opacity may be smooth and rounded, or it may send off radiating shoots into the later-formed peripheral layers of clear cortical. Fig. 77 reproduces the instructive but diagrammatic drawing from

Fig. 77.

Some forms of zonular cataract. (Liebreich.)

[1] Von Ammon, Archiv für Ophthalmologie, iv., 1, Taf. 5.
[2] H. Müller, Gesammelte Schriften, S. 286.
[3] Von Jaeger, Staar und Staar Operationen, 1854, S. 17.

Liebreich. In the first drawing we have a section of an ordinary single-layered zonular cataract, in the second a complete zone of opacity with axial cataract and a partially formed second zone, in the third there are double zones, and in the fourth a poorly developed small lens with a single zone. Fig. 78 (after Spicer[1]) gives a careful drawing of a zonular cataract with some axial opacity, as seen with dilated pupil and oblique light; the space between the inner edge of the pupil and the outer edge of the diseased portion of the lens consists of clear fibres, except where the radiating white lines intrude upon it from the periphery of the cataractous zone. In Fig. 79

FIG. 78.

Zonular cataract. (Spicer.)

FIG. 79.

Right eye. Left eye.

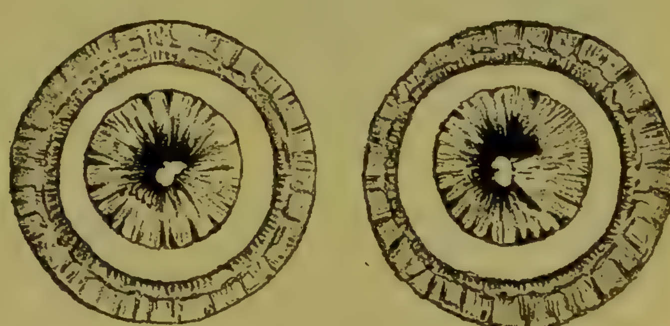
Triple and double zonular cataracts. (Hess.)

(after Hess[2]) are reproduced the mixed zonular and axial cataracts of a woman both of whose children also had congenital cataract. In the right eye we have the representation of a triple zonular cataract and in the left a double one. In each eye the opaque radiating striæ are narrow and pointed towards the pole and broad at their peripheral ends. In each case also the fact that the position of the margin of the lens can be distinctly seen through the iridectomy coloboma shows that it was smaller than normal.

FIG. 80.

Zonular cataract with curved projections into the clear lens-substance. (Jessop.)

FIG. 81.



Zonular cataract with clear central axis. (Heinzel.)

At times, instead of these peripheral radiating fine striæ, we have curved conical projections into the clear lens-substance, the apex of the cone pointing to the centre of the cataract, those in the anterior cortical having the

[1] Spicer, Trans. Ophth. Soc. U. K., 1892, vol. xii. p. 108.
[2] Hess, Archiv für Ophthalmologie, xxxix., 1, S. 182–220.

convexity forward, while those behind the opaque zone have their concavity forward, as is well shown in Fig. 80 (after Jessop[1]). Such are the more usual appearances of zonular cataract, but recently Heinzel[2] has described and pictured a case from Fuchs's clinic where there is a zonular cataract with a clear central axis, as is represented in Fig. 81. According to Purtscher,[3] zonular cataract existed in the upper half of each lens only in a case where there were anterior polar cataract and retained pupillary membrane; there was also interstitial keratitis.

Zonular cataract is often hereditary, but it is the tendency to the formation of cataract rather than the special form of it which is transmitted, and we find at times other forms of cataract develop in other members of the same family. In the vast majority of cases zonular cataract is developed in both eyes and the opaque zone in each eye is of the same diameter. E. v. Jaeger noticed a case where it was monocular, and according to D. E. Mueller[4] we may have in the same individual well-developed zonular cataract on one side with a shrunken cataract (C. arida-siliquata) or even posterior polar cataract on the other. In most instances there is no complication in the shape of atrophy of the optic nerve or disease of the retina or chorioid, and in cases where these are excluded the acuity of vision will vary with the density of the cataract and with the extent to which it covers the pupillary space. In many instances it is sufficient to enable those affected by it, after correction of their astigmatism, to read coarse print with a magnifying glass, and to perform various kinds of manual labor which do not call for accurate eye-sight. When dilatation of the pupil materially betters the acuity of vision, the patient is often much benefited by the performance of a small iridectomy. I have known zonular cataracts to remain unaltered many years, but often as the patients grow older there is an increase in the opacity of the lens and at times a development of senile cataract, as is well shown in the case of Leber,[5] where in typical zonular cataract, six years after the first examination, there was clouding of the entire lens. In other instances the progress of the opacity is very slow, as is well shown in the following case. B. T. was first seen by me in 1871 in his twenty-sixth year. He had in each eye a single-layered zonular cataract measuring as well as could be determined by the ophthalmoscope five millimetres, and stated that he had had defective vision all his life. The eyes were slightly myopic. V. in each without a glass = 20/CC. With —1/36 ⊃ —1/24 Cy. axis 135°, V. = 20/L in O. D.; and in O. S., with the same glass, axis of Cy. at 45° V. = 20/LXX. He wore these glasses for distance, but preferred to read without a glass. Twenty-six years later the patient had become more myopic, and even with a suitable correc-

[1] W H. Jessop, Ophth. Soc. U. K., vol. vii. p. 171.

[2] Heinzel, Deutschmann, Beiträge zur Augenheilkunde, Heft v. S. 27.

[3] Purtscher, Centralblatt für prakt. Augenheilkunde, February, 1894, S. 33–40.

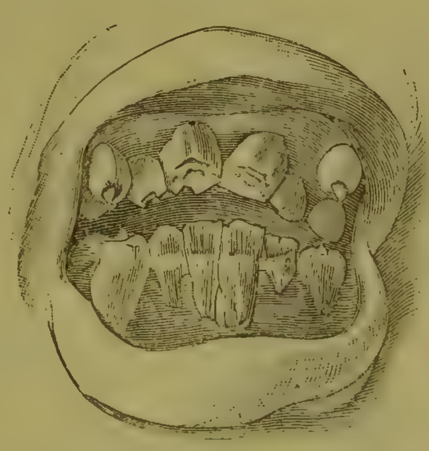
[4] D. E. Mueller, Archiv für Ophthalmologie, ii., 2, S. 166–178.

[5] Leber, ibidem, xxvi., 1, S. 295.

tion vision had become much impaired. The patient stated that this had occurred in the last few years. O. D. —1/7 combined with —1/24 Cy. axis 135°, V. = 20/CC. O. S. —1/7 combined with —1/24 axis 45° V. = 5/CC. The opacities in the lens have become more dense, but the myopia is probably axile, as with dilated pupil a large conus can be seen to the outside of each disk, and in the left eye there were several small spots of chorioidal atrophy in the macular region as well as above and below it.

Zonular cataract may exist in individuals with no marked deviation from general health, but is more frequent in the feeble, poorly-developed, and rhachitic. In the latter class of cases there is frequently also rhachitic deformation of the skull, but more commonly, as Horner has taught us, a coincident development of rhachitic teeth. The central incisors are usually the most affected and present curious irregularities in the distribution of their covering of enamel, and at times in their conformation. The enamel in normal teeth shows a series of fine transverse, almost microscopic ridges, which by reflection of light give the tooth its brilliantly white surface. In rhachitic teeth these ridges are irregularly developed and very much more prominent, with correspondingly wider depressions between them. At the neck of the tooth the enamel, instead of dying out gradually on the roots, ends often with a marked ridge, while the labial and palatine layers of the enamel, instead of making a smooth, even surface at their junction, cause irregular ridges at their point of meeting. According to V. Arx, out of one hundred and eighty-nine cases of zonular cataract sixty-six per cent. had rhachitic teeth. It was at one time the general belief that zonular cataract was formed during adolescence, and Arlt, who pointed out the frequency of hydrocephalic forms of the skull and infantile convulsions in these cases, believed that the violent cramps of the outer and inner muscles of the eyes in such cases were the cause of it. Knies believes that the association of zonular cataract with rhachitis is due to the convulsions so common in rhachitis of the skull, and that the opacity may follow the convulsions within two weeks and that it is due to the cramps of the ciliary muscle. Horner also and many subsequent writers report convulsions during infancy as occurring in a large percentage of the cases of zonular cataract observed by them. Indeed, until Becker published his case, where the zonular cataract was observed by the parents immediately after birth, and where he minutely examined the eyes in the thirteenth week, there was no general belief in the occurrence of zonular cataract during fœtal life. Wecker[1]

FIG. 82.



Irregular and malformed teeth in a case of zonular cataract.

[1] Wecker et Landolt, Traité d'Ophthalmologie, vol. ii. p. 904.

reports a case where in a nine-year-old child at first examination there was well-marked zonular cataract in one eye with an absolutely clear lens in the other. A few months later zonular cataract developed in the sound eye. Graefe reports three cases in which he satisfied himself that zonular cataract developed in previously clear lenses dislocated into the vitreous. Zonu'ar cataract has also been caused in adolescence in consequence of an inflammation due to a perforation of the cornea. Such cases, if it were not for the well-known diagnostic acumen of their reporters, would lead us naturally to think that slight zonular cataract had been overlooked at the first examination and that it had subsequently increased. The refraction of the eyes affected by zonular cataract may be either hypermetropic or myopic, and usually is that of a low-grade myopia. The acuity of vision and the range of accommodation are both diminished. In three cases carefully examined by Becker[1] and Schulek the latter was found to be respectively 1/50, 1/16, and 1/40 of the normal range. The causation of zonular cataract has been more fully discussed under the head of The Pathology of Cataract.

V. **Punctate and Stellar Cataract.**—Liebreich has given us a description of this form of partial congenital cataract where the opacities consisted in exceedingly fine dots so arranged as to show the well-known forms of the lens sectors in the anterior and posterior surfaces of the lens. Hasner[2] places the opacity in the inter-fibrillar material and, as shown in the figure, the rays from the Y in the anterior cortical gave off branches which again divided, although he is uncertain "whether there may not be simultaneous degeneration of the ends of the lens-fibres." He calls attention to the fact that sometimes in senile cataract we find the reverse process, the sectors clouding and the inter-fibrillar substance remaining clear. Zirm[3] gives a case of a boy fourteen years of age where there was hereditary cataract in every generation from the great-grandfather down. There were vast numbers of small dots arranged in stellar form, corresponding to the sectors of the lens, and in some of the largest, Zirm, by means of a magnifying-glass, could demonstrate a clear centre in the individual dots. There was myopia 1/9 and with correction V. = 20/LXX. He also describes a combination of zonular with punctate cataract. Such cataracts are not necessarily found in myopic eyes, and the same author describes one in a hypermetropic eye. Fig. 85 gives an illustration of a punctate stellar cataract, as it appeared when examined with the ophthalmoscope, in a three-year-old child with aniridia who was recently seen in my service in the Hospital of the University of Pennsylvania.

FIG. 83.

Stellar cataract. (Hasner.)

FIG. 84

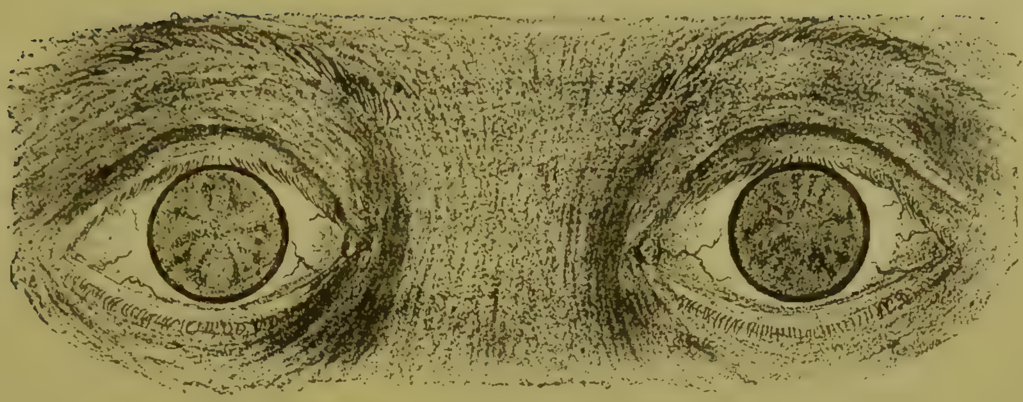
Punctate and stellar cataract. (Zirm.)

[1] Becker, Bericht über die Augen-Klinik der Wiener Universität, 1867, S. 96.

[2] Hasner, Klinische Vorträge, 1865, S 269.

[3] Zirm, Klinische Monatsblätter für Augenheilkunde, 1892, S. 5.

FIG. 85.



Punctate and stellar cataract in a case of aniridia. (Norris.)

VI. **Congenital Central Cataract** (nuclear cataract) is occasionally observed, and is to be differentiated from zonular cataract by its being denser towards the centre, when examined either with the mirror or oblique light. When the rest of the lens is clear the patient may have excellent vision. It is at times combined with other forms of axial cataract, and as in many partial congenital cataracts is not infrequently accompanied by nystagmus. Zirm describes a case with a minute, sharply-cut, ivory-colored central opacity enveloped in a light zonular opacity.

VII. **Congenital Total Cataract**, where it occurs, is due to some form of fœtal disease or malnutrition, and, inasmuch as this may have occurred at any period of fœtal life after the formation of the lens vesicle, we observe such cataracts at birth in very various stages. If they are in their incipiency they often advance very rapidly, and as they become older are usually white in color and soft in consistence, although the capsule and outer cortical layers may be tough and resistant. Becker has proved that a total cataract at birth may be either soft or have gone on to softening of the cortical, with a harder centre dropping to the bottom of the capsule (Morgagnian cataract), or may have become completely fluid with absorption of the denser central portion. Hess [1] has also observed a case of total congenital cataract in a rhachitic child in whose eyes zonular cataracts lay embedded in opaque and softened lens matter, the original lesion having been manifestly the zonular degeneration of the lens. Some such cataracts show even further traces of degeneration, and there remains little but shrunken and opaque capsule with a proliferation of the cells of the anterior epithelium. Becker also relates a most interesting case where a zonular cataract, observed at birth and very well marked in the fourteenth week, had gone on to develop a fluidity of the cortical matter, so that the opaque zonule and nucleus sank in the capsular sac and its anterior surface presented upward. Discission was performed, and in one eye an almost fluid cataractous matter was evacuated which rapidly absorbed, while in the other the more consistent central part of the lens remained and re-

[1] Hess, Archiv für Ophthalmologie, xlvii., 2, S. 309

quired a second operation to secure its absorption. Membranes and shrunken cataracts with adhesions to the iris are more frequent in micro-ophthalmic eyes, and Becker tells us that they are always present in both eyes and usually accompanied by nystagmus, and states that he has often found in them blood-bearing membranes on the outer surface of the posterior capsule, covered by remnants of the embryonal vitreous.

VIII. **The Cataract of Adolescence** often forms very rapidly. The consistence varies. The more it resembles skimmed milk in color the softer it will be found. It differs anatomically from the cataracts of adult life in the fact that the entire lens, including the central portions, undergoes active cataractous degeneration and often becomes entirely fluid. At times it may be infiltrated with lime salts and may become solid throughout. Chalky degeneration is also found in fluid cataracts, which may have considerable quantities of granular cretaceous matter. This tendency to infiltration with lime salts is much greater in the cataracts of youth than in those of old age. The soft cataracts of adolescence go on to shrink, and finally we find a small sac with much thickened walls, just as has been described in the degenerations of congenital cataracts. A certain grade of such shrinking and thickening of the capsule, owing to a fancied resemblance to pod fruits such as peas and beans, has been termed by Adam Schmidt cataracta arida-siliquata—a name which figures in most treatises to the present day. Where cataract develops in adolescents we should always look out for diabetes, for according to most authors the cataract which develops in such subjects presents no clinical characteristics to distinguish it from other varieties of adolescent cataract.

IX. **The Cataract of Mature Life and of Old Age.**—Between the ages of twenty and twenty-five the nucleus of the lens has acquired a yellowish tint, and after the latter period has become quite firm and resisting, so that when cataract forms it requires different treatment from that adapted to the cataract of adolescence. As age advances and the individual approaches forty-eight or fifty the nucleus has enlarged at the expense of the cortical and has become still harder. It is covered only by a thin layer of cortical fibres, the germ force of the epithelium lining the anterior capsule has become almost exhausted and the formation of new fibres very slow, the entire lens has lost elasticity, and is so little capable of responding to the contractions of the ciliary muscle that the patient, if emmetropic, is obliged to put on convex glasses for near fine work. In consequence of the germ force of the epithelial cells being nearly exhausted, but few new lens-fibres are produced, and such as are grow slowly and have all they can do to keep themselves well nourished and normal. Consequently any decided depression of general health or any degeneration of the walls of the vessels of the anterior part of the chorioid, of the ciliary processes, or of the iris may cause the lens to be fed with an imperfect pabulum, and so alter the condition of the soft cortical fibres as to arrest their growth and to cause them to become hazy or even to enter upon a retrograde metamorphosis.

The stoppage of growth is accompanied by a diminution of intracapsular pressure, and the nutritive fluids in the lens, together with any products of decomposition of the cortical fibres, find their way between the fibres. If such a state of malnutrition exists in the cortical, a somewhat similar state of affairs sets in in the nucleus, but to a vastly less extent. The fibres having become dense, hard, and horny, endosmosis is slow in them, and any breaking down of tissue takes place to a very limited extent. The normal increase of density of the nucleus is readily appreciated when, after dilatation of the pupil, we examine the lens with the ophthalmoscope with a magnifying-glass behind the mirror. Then by motions of the mirror we can perceive the outlines of the nucleus differentiated from the less dense cortical. When, in addition to the increasing density of the nucleus with advancing years, cataractous degeneration begins in the lens, we can readily see its first indications in the form of black streaks or cones, arranged in the anterior and posterior cortical near the equator of the lens, with or without similar ones at the equator of the nucleus. Attentive examination will often show the simultaneous occurrence of very fine dots or opacities in the perinuclear cortical. More rarely the perinuclear opacities are the dominating feature, and we then have so-called nuclear cataract. All these changes are usually slow to increase, and where they are mostly limited to the peripheral cortical they often exist for years without giving the patient any considerable inconvenience. In the vast majority of cases these first changes in the peripheral cortical are most developed in the inner lower quadrant of the lens and often precede their appearance in other parts of the periphery. When we examine these appearances with a magnifying-glass and oblique light the dark streaks and cones and dots appear gray and whitish and any perinuclear haze grayish. By this latter method of examination we also obtain a yellowish reflex from the nucleus. In some over-ripe cataracts with very large and dense nucleus this reflex may appear so reddish as to make the beginner think he is getting red light reflected from the fundus. In the incipient stage the pupil to ordinary inspection appears less black than usual, with at times a yellowish-gray tint. As the cataract advances the lens swells, the anterior chamber becomes narrower, and the pupil gradually grayer. The anterior layers of the lens often assume a peculiar glittering reflex, at times resembling mother-of-pearl. Later the pupil appears yellower, the lens becomes less swollen, the anterior fibres lose their glittering aspect and assume a more or less transparent yellow look. At times the intersectorial lines appear dark and the sectors themselves gray, but usually as the cataract becomes older the intersectorial lines appear whitish. When the lens becomes less swollen, and when in addition to the appearances above described the anterior chamber has resumed its normal depth, the cataract is said to have become ripe. At this stage the opacity of the anterior cortical has often become so great that the posterior pupillary surface of the iris appears to lie immediately against it, the capsule being so thin and transparent that its presence is not recognized,

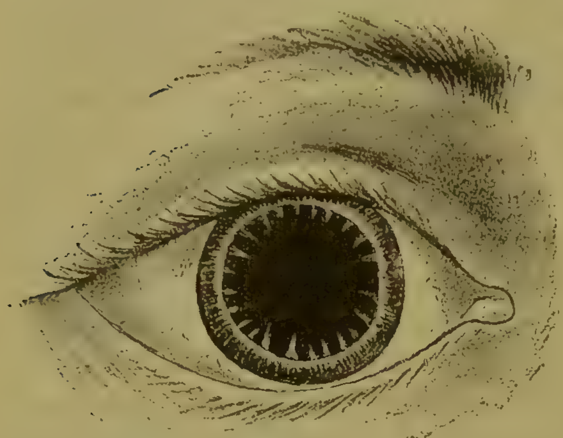
while in the earlier stages the opacity of the anterior cortical is almost entirely some distance behind the iris, and there then appears to be a clear dark space between the posterior surface of the iris and the cataract. As time goes on, opacities form also in the capsule and its epithelium as very fine whitish dots or streaks, and by careful examination with oblique light they often look as if they were running inward into the lens-fibres. In some very old people the nucleus grows to occupy almost the entire lens, leaving a very slight layer of cortical. In such instances the pupil remains comparatively dark, and the cataract, when extracted and held against the light, appears of a dark reddish-brown hue. Such cases are termed black cataract. Opaque lenses may also become black from absorption of the products of decomposing blood where there has been a previous hemorrhage into the vitreous or into the anterior chamber. The subjective complaints of the patients suffering with forming cataracts are, in the earlier stages, of muscæ, monocular polyopia, indistinctness of fine objects, and ready fatigue of the eye. Later we have a gradually increasing gray fog enveloping and hiding all objects and giving the brightest sunlight a moonlight pallor. The attitude of patients with moderately developed cataract is that of looking down and groping, for they still have perception of light, and by thus shading the eyes and dilating the pupil try to obtain brighter retinal images; and this position is in marked contrast with the erect gait and fixed look of those who have amblyopia due to atrophy of the optic nerve. In incipient cataract, where it is peripheral, the patients seek a strong light, trying thus to see through the fog in front of them, while in nuclear opacity the eye is shaded and the back turned to the light to obtain vision through the wider pupil and the comparatively clear periphery of the lens. In fact, most cataract patients find comfort in the comparatively large pupil which is obtained in moderate illumination, and welcome the greater amount of light thus admitted to the eye. In the stage of swelling of the lens a strong light is almost always uncomfortable, owing probably to the irritability of the iris and ciliary body caused by the pressure of the swollen lens. As has already been dwelt on under the head of "Diagnosis," every cataract case should have good central and peripheral light perception. As time goes on further degenerative changes take place in cataractous lenses; ripeness is succeeded by over-ripeness, capsular opacities form, or, if they have existed, become more marked and numerous. The cortical layers again become slightly more transparent, the lens shrinks and often wabbles slightly with the sudden motions of the eye, and the patient sees rather more clearly. At times, however, another form of degeneration of the cortical fibres takes place; they form a semi-fluid or fluid whitish mass and allow the still firm and yellow nucleus to fall to the bottom of the capsular sac and often to come into view when the head is tilted forward. This form of lenticular disease is termed Morgagnian cataract. The further changes which at times occur in cataractous eyes are discussed more fully under the head of "Second Sight." The refraction of the eye seems to have but little influence on the

development of cataract, although myopic eyes appear to have more frequently a slowly developing form of nuclear cataract. In the vast majority of cases there is hypermetropia, but it is to be remembered that even in the educated classes this is the prevailing state of refraction, and it is therefore more likely that the majority of cases will be found in such eyes. The swelling of the lens is accompanied by a proportionate amount of increase in the refraction, but there is usually sufficient accompanying cloudiness to prevent the patient experiencing any advantage from it in his near work. There is, of course, irregular swelling of the sectors which produces a monocular polyopia and much irregular astigmatism, but we also often find that a reversal of the normal astigmatism has occurred, and that the horizontal meridian has acquired the highest refraction. Very often, however, even when sight for distance is markedly improved by a proper cylindrical correction, the vision for reading is not correspondingly benefited by it. While it is in the experience of every surgeon that the general health has a decided influence on the development of cataract, and we constantly see patients with marked and rapidly increasing clouding of the lens coming on immediately after an attack of pneumonia or low fever, and where the sickness has either originated the cataract, or, if previously existent, has pushed it on to much more rapid development, nevertheless we have so far not been able to define the exact pathological states in the eye or general system which bring this about. *Disease of the kidneys* is said to predispose to cataract, and Deutschmann has introduced the term *cataracta nephritica*, and from the examination of a series of cases as to the frequency of the occurrence of albumin in the urine of cataractous patients finds that thirty-three per cent. of those so affected are albuminuric; while Becker gives in a first series two per cent. and in a second eighteen per cent. The author's own experience is that few of those who have presented themselves to him for cataract operation have had albumin in the urine, and in the majority of cases where it was present it was evidently due to prostatic and vesical affections, casts of the uriniferous tubules being only exceptionally found. On the other hand, he has examined and carefully recorded the appearances of the eye-grounds of over a hundred cases of Bright's disease in the wards of a general hospital, and in many of those who were dropsical and pallid, with difficulty of breathing, who, in a word, presented the last stage of the affection, he found incipient cataract. Diabetes mellitus probably gives a higher percentage of cataract in the young than any disease with which we are acquainted, but most authorities agree that the form of its development has nothing characteristic, although Foerster, while admitting that cataract in diabetics in middle life develops as do other forms of senile cataract, claimed for the young a peculiar milky, glistening appearance immediately under the anterior capsule as being characteristic. Further observation, however, has shown that this mode of development is probably not the rule. (Horner, Becker, Leber.)

Senile cataract invariably attacks both eyes, but usually not quite simul-

taneously, one eye being attacked some time later than its fellow. While, as has just been mentioned, the general health has much to do with formation of cataract or the hastening of its progress where already formed, it is very exceptional for cataractous processes to become regressive, and for opacities when once formed to diminish in density or to disappear entirely, although it is within the experience of every practitioner that they may be stationary for a long period of time. Seegen, Tannahill, and Koenig, as already mentioned in a previous section, have observed the spontaneous absorption of diabetic cataract. Jaeger reports the case of a young myope where there were marked equatorial striæ in both the anterior and posterior cortical, which decreased gradually during a period of two years, and which in five years had entirely disappeared. Fig. 86 gives a reproduction of his drawing in this case. Jaeger, however, remarks that, while he has often seen such manifestations become stationary in traumatic cataract and in cataract due to slight chorioiditis, he never under any circumstances observed the clearing up of even the slightest senile opacity. Fig. 87, also from Jaeger, gives a view of a marked stellar posterior cortical cataract with fine striæ in the same layer of the lens, and with slight peripheral opacities in the anterior cortical, which remained stationary for twelve years. The eye in which this developed was emmetropic.

FIG. 86.



Equatorial striæ in eye of a young myope which cleared up entirely in five years. (Jaeger.)

FIG. 87.

Stellar posterior cortical cataract which remained stationary for twelve years. (Jaeger.)

*The Cataract of Extreme Old Age.*—The anatomical characteristics of cataract in extreme old age are essentially those of ordinary cataract, although, owing to the fact that the nucleus is larger and that the cortical layers are thinner, we have a harder cataract. There is usually a development of arcus senilis, an increased rigidity of sclerotic, and at times an atrophy of the orbital fat, which gives the eyeball a deeper position in the orbit. Where, however, the nutrition is fairly good, operations on patients between eighty and ninety still offer fair prospects of success, and the prognosis will vary in individual cases. It must also be borne in mind that the reparative processes and assimilative powers in some patients at eighty are still quite

as good as in those of the average individual at seventy or seventy-five. Higgens, in giving the results of nine hundred and twenty-five cataract operations, records nineteen cases in which the patients were over eighty years of age. Of these, seventeen were successes, one a partial success, and one a failure. H. Derby[1] reports thirty-four cases in individuals between eighty and ninety, the oldest being eighty-nine. Of these, twenty-nine were successes, two partial successes, two failures, and one was unrecorded.

### HEREDITY.

Another not uncommon cause of cataract is heredity. This transmission from one generation to another may develop during fœtal life and be demonstrable in the offspring at birth, or the tendency to degeneration may manifest itself in later life. In any case the tendency is transmitted, but it does not at all follow that the same form of cataract will develop. In this, as in many other transmitted defects, we see occasionally the phenomenon of atavism, or breeding back to a more remote ancestor, especially to the grandparents, the intervening generation being apparently healthy. As is well known, heredity is frequently shown in the transmission of other eye-diseases, as in typical pigment degeneration of the retina, in hereditary atrophy of the optic nerves, and in color-blindness. Farre,[2] editing Saunders's posthumous work on Congenital Cataract, gives the history of four families in each of which several members had this affection. Mackenzie[3] has seen several members of one family either born with cataract or acquiring it at a given age. Arlt[4] saw a brother and two sisters who became cataractous when about twenty years of age. This author quotes Beer and Lusardi as giving good instances of heredity. S. S. Dyer[5] records an instance in which cataract was transmitted to all male members of the family for three generations, while only some of the female members were affected. Hirschberg[6] reports a case of cataract occurring in an individual of thirty years of age, whose four children became affected about the same time of life, and whose grandchild had zonular cataract. Baudon[7] gives the following history of a cataractous individual who had seven children. Of these, five were affected, and of these five, three married. No. 1 had four children, three of whom had congenital cataract; No. 2 had four children, of whom three also had congenital cataract; while No. 3 had only one child, whose eyes were healthy. Berry[8] records a history of hereditary cataract extending through five generations and affecting fifty-five members of the family. J. Tatham

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[1] Hasket Derby, Boston Med. and Surg. Journ., 1895, vol. cxxxiii., No. 13, p. 319.
[2] Farre, Saunders, Diseases of the Eye, London, 1811, p. 134.
[3] Mackenzie, Diseases of the Eye, 1854, p. 747.
[4] Arlt, Die Krankheiten des Auges, 1863, S. 292.
[5] S. S. Dyer, Provincial Med. and Surg. Journ., August 19, 1846.
[6] Hirschberg, Nagel, Jahresbericht, 1874, S. 255.
[7] Baudon, Recueil de Mém. de Méd.. 1887, xxxiii. p 646.
[8] Berry, Ophthalmic Review, 1888, v 1 vii pp. 1–6.

Thompson[1] observed a family where cataract was hereditary for four generations. It usually developed in early childhood, being noted in many instances between three and five years of age. Green[2] reports twenty-one cases of cataract out of seventy individuals in one family. Galezowski[3] gives an analysis of four thousand seven hundred and seventy-six cases of cataract, and concludes that in about four and a half per cent. heredity plays a part in their development. Fukala[4] also gives an interesting series of cases: A cataractous grandmother had thirteen children, of whom nine were cataractous and five transmitted the tendency to their children (three females and two males). One daughter had five children, three of whom had cataract; a second daughter had two cataractous sons out of a family of ten children; the third daughter, out of a family of four, had three cataractous children. The two sons had each four children, and out of these each had one female child affected with cataract. In this family the cataracts developed during adolescence. Wray[5] reports six cases of disseminate dot cataract in the children of one family, in which examination showed a similar condition of the mother.

Fromaget[6] relates the occurrence of hereditary cataract in six generations of one family, three of the cases which he himself examined proving to be zonular cataract. Becker[7] operated for cataract on a woman whose mother and grandmother had previously been operated on successfully by other practitioners. A curious case of hereditary tendency is reported by Dixon, where cataract developed simultaneously in twin brothers at the age of fifty-four, in whom there was an extraordinary resemblance of size, shape, features, distribution of hair, as also of the contours and extent of the bald spots on the cranium. The cataract in each instance was much more advanced in the left eye than in the right, and ripened and was successfully extracted at the same time.

## TRAUMATIC CATARACT FROM CONCUSSION WITHOUT RUPTURE OF THE CAPSULE.

Quite a large number of cases of cataract are on record where, after blows on the eye or in its vicinity, the lens becomes opaque without any visible rupture of the capsule, but so long as no such case has come to autopsy, and we are familiar with ruptures of the posterior capsule, they are always open to the suspicion that there has been a rupture at some point not accessible to examination. Berlin[8] (as previously mentioned) found that by striking the corneæ of rabbits' eyes with an elastic rod he not only

[1] J. Tatham Thompson, Trans. Ophth. Soc. U. K., 1890, vol. x. pp. 141–145.
[2] Green, J., Trans. Amer. Ophth. Soc., 1891.
[3] Galezowski, Recueil d'Ophtalmologie, 1883, p. 17.
[4] Fukala, Nagel, Jahresbericht, 1890.
[5] Wray, Trans. Ophth. Soc. U. K., 1892, vol. xii. p. 109.
[6] Fromaget, Gazette hebd des Sciences Méd. de Bordeaux, 1893, No. 31.
[7] Becker, Graefe und Saemisch, Handbuch, Bd. v. S. 262.
[8] Berlin, Klinische Monatsblätter für Augenheilkunde, 1878, S. 42–78.

produced abrasion of the corneal epithelium with opacity of that membrane, but also a clouding of the anterior cortical of the lens, commencing opposite the point of injury of the cornea and often spreading to a considerable extent. Deutschmann[1] has reported two cases of partial transient opacity of the lens in wounds of the sclerotic at the periphery of the anterior chamber. After excision of the prolapsed iris a diffuse opacity of the lens was visible immediately beneath the capsule, with some whitish streaks and points, the line separating the opacity from the clear portion of the lens being sharp-cut. Under rest and a pressure bandage the opacity entirely disappeared in thirty-six hours. Becker[2] has given a well-observed case of cataract from concussion without rupture of the capsule. The eye received a blow from a falling icicle, which did not rupture the external coat but caused a rent in the chorioid which was distinctly visible through the then clear lens. Three weeks later there was an opacity of the lens about the size of a moderately dilated pupil. The same author also describes a transient opacity of the lens after an iridectomy for glaucoma, where the lens subsequently cleared entirely and where no rent in the capsule was demonstrable. He attributes the opacity to pressure on the anterior surface of the lens at the time of the evacuation of the aqueous.

### TRAUMATIC CATARACT CAUSED BY RUPTURE OF THE CAPSULE.

On the other hand, the cases of transverse fracture of the anterior capsule caused by hanging, reported by Dyer[3] in 1866, show how cautious we ought to be in drawing conclusions that injuries at a distance from the eye may not after all cause cataract by capsular rupture. In Dyer's case the drop was three feet and the examination thirty-five minutes after death. In both eyes the rent in the capsule ran parallel with and about a line below the horizontal meridian of the lens. In the right eye it looked like a crack in clear ice, while the whole lens was iridescent and a fissure in it ran back to the centre, whence it was continued by several little lines projecting backward. In the left eye, the crack he thought was confined to the capsule, but had a feathery look from lines running off from it upward and downward. In three dogs which he hung the lens was fractured in two only. Green[4] has since shown that it is not a necessary lesion from hanging; in the executed criminal examined by him it was not present. Robert[5] gives an instance where the spine of a chestnut burr penetrated the cornea and lens-capsule. The spine was removed through the corneal wound. The crystalline presented a milky appearance which subsequently cleared up. Cuts and ruptures of the capsule usually cause cataract by admission of

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[1] Deutschmann, Untersuchung zur Pathogenese des Kataracts.
[2] Becker, Graefe und Saemisch, Handbuch, Bd. v. S. 275.
[3] Ezra Dyer, Transactions of the Amer. Ophthal. Soc., 1866, p. 13.
[4] Green, ibidem, 1875, p. 354.
[5] Robert, Annales d'Oculistique, 1856, p. 127.

aqueous to the lens-substance, and in most instances as the lens-substance swells it enlarges the rent, admits more aqueous, and causes eventual total opacity of the lens. This, however, is not always the case, for minute and sharp-cut wounds in the capsule may heal and the cataract clear up or remain partial. Thus, Fischer reports a case where he did a discission on both eyes of a child, and that the capsules were really opened was proved by the escape of a milky fluid into the aqueous. A few days later, the child dying from some intercurrent disease, Bochdalek after careful examination was unable to find any rupture or cut in the capsules. Occasionally the scars from incision in the capsule are demonstrable, the lens remaining clear. Mackenzie[1] observed a scratch on the anterior capsule where a permanent white mark remained without resultant opacity of the lens. Bresgen reports two cases where the rent in the capsule became covered with yellowish exudation, which finally cleared up, as did also the slight underlying cortical opacity, but more frequently where healing takes place it is after the extrusion of a small mass of lens-matter through the minute wound. Landesberg describes a rupture of the anterior capsule from a blow with the flat hand, which extended for a distance of six millimetres. A good deal of clouding of the anterior cortical followed, but the capsular rent healed, and the pupillary space cleared up with full acuity of vision. Liebrecht also reports an extensive rent in the anterior capsule from a non-penetrating blow with a piece of iron. There was considerable gaping of the middle of the wound, and the capsule was thrown into folds running at right angles to the rent. The capsular wound soon presented a delicate streaked cloudiness, the edges becoming thicker and whiter, and gradually closed. Four weeks after the injury there was a faint streak two millimetres wide in the position of the rupture, and some months later V. = 5/14 and Sn. III could with difficulty be read. In some instances where a foreign body penetrates the lens and remains caught in the wound it may fit so tightly as for a time to exclude the aqueous and prevent any considerable opacity of the lens, which may, however, follow later as the little cleft enlarges. This is well exemplified in a case of Forlarini,[2] where a steel splinter filled the gap so completely that for a month the lens remained clear, and then, owing to admission of aqueous at the edges, gradually clouded. In some rare cases it would appear as if the lens-capsule presented a rare degree of elasticity and closed entirely on the track of the foreign body. Thus, Purtscher[3] records a case of penetrating wound of the lens by an iron splinter, where sixteen years later the track of the foreign body was visible and clouded, but the lens was otherwise clear. Doyne[4] gives a still more remarkable case where a steel splinter which perforated the eye had lodged in the retina. Scars were demonstrable in the

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[1] Mackenzie, Diseases of the Eye, 4th edition, 1854, p. 397.

[2] Forlarini, Nagel, Jahresbericht, 1872, p. 408.

[3] Purtscher, Centralblatt für prakt. Augenheilkunde, 1881, S. 161.

[4] R. W. Doyne, Trans. Ophth. Soc. U. K., vol. x. p. 198.

cornea, in the anterior and posterior surfaces of the lens, as well as the track in the vitreous, the little particle of steel partly enveloped in a blood-clot lying just below the disk. The vision was 6/60. One month later the lens was still clear and the foreign body still visible in the retina. Desmarres, Jr., also relates a case where the lens clouded temporarily from a foreign body entering and lodging in it, the opacity subsequently clearing up and leaving only a small black spot surrounded by a very limited opaque area. Jackson and Schneidemann report a case where the perforating foreign body left a track in the cornea, iris, and lens and where it could be seen lodged in the retina, the vision still remaining 5/7. As an instance of the occasional slow clouding of the lens after a large incision into it may be quoted the case related by the same authors, where a free discission in the lower part of a congenitally dislocated lens caused local opacity with subsequent clouding and absorption, leaving a notch in the lens, while the upper part remained clear. It required a subsequent operation to cause the absorption of the upper part of the lens. Milliken, Ayers, Aschmann, Eugene Smith, and Lippincott have all reported somewhat similar cases of partial clouding after injury. The longest period for which I have ever known the lens to remain clear with a foreign body embedded in it is twenty-eight years. In 1895 I saw, in consultation with Dr. G. C. Harlan, a man one of whose eyes had been destroyed by a blast twenty-eight years previously. The other eye had been injured at the same time, and at the time of examination presented a whitish track in the centre of the lens with a few granular opacities in the anterior cortical and a semilunar patch of opacity in the posterior cortical up and out. The track penetrated the lens for about half its thickness, and at the bottom of it lay what appeared to be a glittering particle of mica. The vision was 20/CC, and for all these years he had been able to perform coarse labor, but two years later (1897) his sight was becoming dimmer. Small foreign bodies, such as minute splinters of steel from hammers, not infrequently perforate the lens and lodge in it. They usually, however, cause total opacity, and it is very difficult to demonstrate their presence unless they lie very superficially. After long sojourn they may more or less completely undergo oxidation, and the minute particles of rust are carried by the osmotic current to various parts of the lens. Some authors then speak of *siderosis* of the lens. Iwanoff long ago called attention to the fact that in wounds of the lens fragments of copper driven into it seemed more irritating than similar pieces of iron. This is probably due to the chemical action of the copper salts formed by the action of the intra-ocular fluids on this metal, and is made more probable by the fact that, as Leber has shown, any metals in fine aseptic powder introduced into the vitreous cause deleterious changes in the eye, the eminently insoluble ones, like gold and silver, acting more slowly by reason of their slow solution. Doyne,[1] however, reports a case where a

[1] Doyne, Trans. Ophth. Soc. U. K., 1894, vol. xiv. p. 219.

fragment of gun cap lodged in the inner posterior part of the lens and where the lens remained so transparent for twenty-eight years that the patient, a soldier, could aim and shoot with this eye. At this date the lens became so cloudy that he could no longer use the eye in this way, but still at the time of examination the fragment of cap could be discerned, thirty years having elapsed since the time of the accident. Probably the most remarkable case of the sojourn of a comparatively large body in the lens is that recorded by Pamard,[1] where a bird-shot lodged in the posterior layers of the lens. The inflammatory symptoms partially subsided, but one month after the injury the recurrence of severe pain and insomnia caused an operation of extraction to be performed, the bird-shot coming away with the lens. The result was a black pupil but no useful vision.

### RUPTURE OF THE POSTERIOR CAPSULE.

Cataracts produced by the rupture of the posterior capsule are not usually demonstrable except when accident gives us an opportunity to anatomically examine the eye, but White Cooper[2] saw a man who had received a severe blow from a stick over the nose and angle of the eye, and who complained two days later of monocular double vision and of cloudy vision, in whom slight haze of the lens and a sharp-cut cloudy line on the posterior capsule were demonstrable. Treacher Collins[3] pictures a case where rupture of the posterior capsule was demonstrated by autopsy, and the figure which he gives is here reproduced. The eye received a blow from a stone and was enucleated a week later. Owing to the weak solvent action of the vitreous the anterior portion of the lens was still clear. Wounds of the lens are always serious, and in most instances, even where the track of the wound is directly through the pupillary space, and where the cornea and lens are the only parts of the eye injured, lead to total opacity of the lens. At times a very minute puncture will suffice to cause complete cataract. I recently saw a case where a negro child about ten years of age had accidentally stabbed itself in the eye with a fine sewing-needle. There was a very minute dot of opacity almost in the centre of the pupil, but a flocculus of lens-matter soon protruded, to be followed by others until the entire lens became opaque and was in the course of a few months entirely

Fig. 88.

Traumatic rupture of the posterior capsule. (E. Treacher Collins.)

[1] Pamard, Annales d'Oculistique, 1860, t xliii. p. 23–25.
[2] White Cooper, Wounds and Injuries of the Eye, p. 176.
[3] E. Treacher Collins, Trans. Ophth. Soc. U. K., vol. xi. p. 126.

absorbed. The pupil was kept dilated by atropia, and there was scarcely any irritation of the eye during the progress of the case, and after the absorption of the lens there was excellent vision with a cataract glass. In penetrating wounds where the iris is perforated by the instrument the exudation of lymph and the formation of a synechia tend to close the capsular wound and prevent the formation of total cataract. In cases where the wound is larger this protective influence of the iris and effused lymph is a great disadvantage, excluding the aqueous from the totally opaque lens and causing a permanent cataract. Moreover, the involvement of the iris from the inflammation which ensues, and its tendency to spread backward to the ciliary body and chorioid, is always a serious complication, and the mere fact of the congestion of the vessels around the periphery of the cornea so interferes with exosmosis that the solution of lens-matter by the aqueous is always slow during its continuance. Deep wounds of the ciliary region, involving the lens, are among the most dangerous and treacherous wounds of the eye, frequently giving rise to inflammation so severe that the eyeball gradually shrinks, and only too often to sympathetic disease of the fellow-eye. Where such wounds are made with unclean foreign bodies, missiles, or instruments they often lead to suppuration of the eyeball. It is astonishing, when we consider how much care it requires to keep our surgical instruments sterile, how frequently small chips of iron and steel driven into the eye seem to carry little or no infecting material with them.

## CAPSULAR CATARACTS.

By this term we designate any opacity of the capsule or any mass formed on its inner side by the proliferation of its epithelial cells. The white spots occurring in over-ripe cataracts have already been described, and besides these we encounter more extensive changes, due usually primarily to some inflammation of the uveal tract, and very frequently to exudation gluing extensive surfaces of the iris to the anterior capsule. Under such adhesions the capsular cells proliferate and form a mass projecting into and displacing the lens-fibres, and by this action, as well as by interference with the formation of new fibres from the perverted activity of the epithelium, we have an opacity formed primarily under the affected part, but which often eventually causes the whole mass to become opaque. The cataracts which form after an irido-chorioiditis, detachment of the retina, etc., are usually of this nature, and in them we find growth and subsequent degeneration of the cells of the anterior capsule, and often also a newly-formed epithelium lining the posterior capsule. One variety of capsular cataract—viz., the *acquired anterior polar*—has always attracted much attention on account of its singular form and position and the fact that the lens-substance often remains clear in other parts.

## ACQUIRED CENTRAL ANTERIOR POLAR CATARACT.

Congenital anterior polar cataract has already been described. The acquired form has been proved by the accurate clinical observations of Arlt to be caused by perforating ulcers of the cornea, where the lens, uncovered by the iris, comes in contact with a corneal ulcer, and the anterior capsule becomes covered with exuded lymph. The underlying epithelium proliferates, and forms a cone projecting into the lens substance, and this is at times surmounted by a thread or by an outwardly-pointing cone of lymph, running to the seat of the previous corneal ulcer. This thread or cone may persist after the closing of the wound has restored the intra-ocular pressure and allowed the aqueous to push the lens back into its place. Usually, however, this filament is ruptured, and we find a pyramid with its apex outward, or at times a flattened and irregular mass of opacity. According to Arlt, where the whitish, projecting mass is over two millimetres in thickness it is usually pyramidal in form. A portion of the pupillary margin of the iris is often adherent to the cornea, but very rarely is there any iris pigment in the projecting mass of cells on the anterior surface of the capsule. The capsular opacity is stationary, and folds of the capsule are often seen projecting as radii from it. The lens-substance beneath the capsular cataract is also cataractous, while the rest of the lens is usually transparent, although occasionally there is complete opacity accompanying it. Anterior central capsular cataract is usually a sequence of the ophthalmia of the new-born. It seldom develops in childhood, never in later life. Knies[1] had an opportunity of observing such a cataract in its early stage while the newly-formed tissue was still transparent. Schweigger[2] has proved that the corneal ulcer need not be accurately central in order to produce anterior polar cataract, and infers that the pupillary capsule undergoes usure and thinning from the pus in contact with it, while the remaining capsule is protected by the iris. Hulke[3] has shown that in the shallow anterior chamber existing in the new-born an exudation of lymph may produce pyramidal cataract without any perforating corneal ulcer.

## THE CAPSULAR CATARACT WHICH FOLLOWS THE REMOVAL OF THE LENS. (SECONDARY CATARACT.)

In all cases of extraction in which the capsule is not removed from the eye with the lens, in all operations by discission, and in many cases of reclination where the operator does not succeed in dislocating the capsule of the lens into the vitreous humor, there remains behind, in the position formerly occupied by the lens, enough capsular remnants to constitute a membrane, which, however transparent at the time of the operation, is likely subsequently, by thickening and folding, to become a hinderance to

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[1] Knies, Klinische Monatsblätter für Augenheilkunde, 1880, S. 181.

[2] Schweigger, Archiv für Ophthalmologie, xxvi., 1, S. 134.

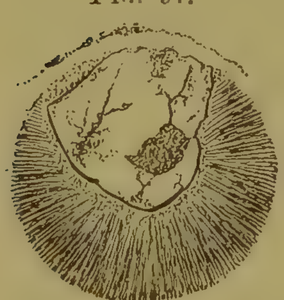
[3] Hulke, Royal London Ophthal. Hosp. Reports, vol. i. p. 189.

vision. In cases of normal extraction, the torn anterior capsule folds and retracts in such a way that it leaves the pupillary space free and becomes adherent to the posterior capsule farther towards the periphery, the anterior and posterior capsules thus enclosing a space in which lie remnants of lens-matter and epithelium of the anterior capsule. This extends in a more or less regular ring behind the insertion of the iris. The closing in of this space and the adherence of the anterior to the posterior capsule exclude the aqueous humor and prevent its action on the remnants of the cataractous lens and of the epithelium which are left behind in this position; and the epithelium goes on to form large nucleated cells and, at times, new but imperfect lens-fibres, which permeate and enclose the remnants of cataractous lens-matter remaining in the little sac, and by this growth form a mass which is more or less transparent and is larger, usually, in the eyes of young individuals, where a greater amount of unused germ force lies dormant in the epithelial cells. In the young these masses of proliferating epithelium enclosed between the two leaflets of the capsule may attain considerable size, and they often exhibit curious forms. This is well shown by the case of Snell,[1] where the lens was removed by a vectis and the operation gave good visual acuity. Six years later the patient noticed a pear-shaped body in front of the pupil which gradually increased in size until it equalled half the diameter of the cornea. It was removed, and examination by Treacher Collins showed it to be lens-capsule covering degenerate lens material. After its removal the patient had V. = 6/6. So long as the new formations stop at this point, being hidden behind the iris, they exercise no untoward influence on vision, but very commonly within a few months after the operation we recognize some thickening of the posterior capsule in the pupillary space as well as of any bands of torn anterior capsule which cross it, and a corresponding blurring of the retinal image is produced. Indeed, even immediately after an operation, as soon as the eye has become quiet enough to allow the patient to use it, the ophthalmoscope will usually show that the pupillary space is not everywhere equally clear, but that only portions of it are perfectly transparent, and the patient soon finds that he cannot see equally well through every portion of it, but that in some positions of the eye and head he sees much better than in others. In fact, he has (so to speak) to choose the hole through which he desires to see, and to adjust either his eye or the object looked at so that it shall come into the line of vision. Such capsular pupils, if so they may be termed, may at times contract and thicken decidedly from low-grade inflammatory processes soon after the operation, or they may remain transparent for years, but more commonly at varying periods will commence to thicken and close, so that often in from six months to two years after an operation the patients will find that their vision has again so far failed that they once more apply to the surgeon for relief. In those cases where in-

[1] Snell, Ophthalmic Review, 1893, p. 345.

flammation sets in after cataract operations, whether it be of low or high grade, we often have at once sufficient thickening of the capsule to render the results of the operation nugatory and to make a capsulotomy necessary. Late-forming capsular opacities often develop without any assignable cause, while at other times they seem to be brought on by some accidental slight inflammation, such as a slight catarrhal conjunctivitis, or by the congestion of the anterior part of the eye, which normally occurs after prolonged use of it for near work. Where these capsular opacities are slight and are not complicated by any morbid process in the vitreous, ciliary processes, or iris, they are readily torn or cut by the knife or needle, a loop of vitreous comes forward into the rip in the capsule, and the vision often remains clear for the remainder of the lifetime of the individual. When, however, these processes of capsulitis are complicated by adherent synechiæ, by any inflammation of the iris, chorioid, or ciliary body, or by vitreous disease, we have a state of affairs much more difficult to remedy, and which often baffles entirely the resources of the surgeon. Of the local causes which produce such secondary cataract some of the most frequent are the healing of a piece of capsule or a part of the iris in the extraction wound. A study of the various forms of secondary cataract is interesting to every surgeon, and nothing can more thoroughly convince him that any of our present methods of cutting or tearing the lens-capsule are imperfect than the utter want of uniformity of his results as regards the size and position of the aperture in the capsule which results from his best efforts to secure a central hole. The following figures, taken from cases which were practical successes, will give some idea of the various forms of secondary cataract complicated with slight adhesions of the capsule to the iris. Fig. 89 was taken about a year after the operation. The patient was a negro, which perhaps accounts for the dense pigmentation of the capsule. With $+ 1/3\frac{1}{2}$ combined with $+ 1/42$ Cy. axis 105°, V. = 20/XL, there being a free clear space at the inner nasal part of the pupil, and the vision being good and eye quiet, in spite of a probable incarceration of the capsule in the wound. In Fig. 90 the more useful inner and lower part of the pupil has a spider-web opacity, while an oblique ovoid rent remains clear. Fig. 91 shows an incarceration of the iris on one side of the wound with several masses of densely opaque spots in the capsule in the immediate neighborhood of slight pigment synechiæ. Fig. 92 shows a dense branching opacity covering most of the pupillary space and starting from a slight adhesion between the iris and the capsule. Fig. 93 shows an ovoid pupil with secondary cataract occupying the lower inner and outer parts; the eye had been myopic, with patches of chorioiditis, and the drawing was taken three weeks after extraction of the cataract. With + 1/15 Cy. | — 1/15 Cy. axis 90°, V. = 20/CC. Fig. 94 shows

FIG. 91.



Secondary cataract after extraction with iridectomy. (Drawn by Dr. B. Alexander Randall.)

Fig. 89

Fig. 90.

Secondary cataract after extraction with iridectomy.
(Drawn by Dr. B. Alexander Randall.)

Fig. 94.

Fig. 95.

Secondary cataract after extraction with iridectomy.
(Drawn by Dr. B. Alexander Randall.)

Fig. 96.

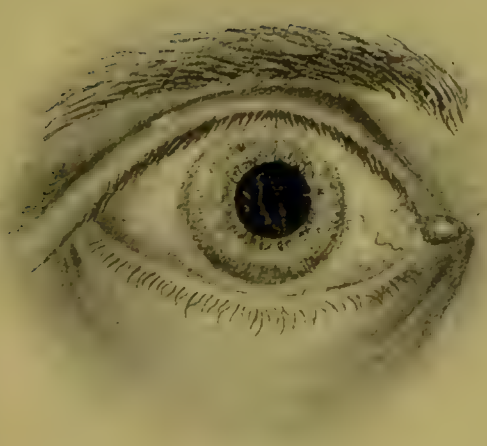


Fig. 97.

Secondary cataract after simple extraction.
(Drawn by Margaretta Washington.)

a clear pupil so displaced upward by the cicatrizing processes as materially to diminish its usefulness to the patient. With head and eye in favorable position + 1/4 ⊂ + 1/9 Cy. axis 150°, V. = 20/C. Fig. 95 shows a case of complicate cataract where there is a curious detachment and upward displacement of large portions of the uveal coat of the iris, which adheres to the capsule. With + 1/3 ⊂ + 1/24 Cy. axis 180° V. = 20/XXX.

The foregoing illustrations have all been taken from cases where a broad iridectomy had been made at the time of extraction because the large

FIG. 92.

Secondary cataract after extraction with iridectomy.

FIG. 93.

Secondary cataract after extraction with iridectomy.

(Drawn by Dr. B. Alexander Randall.)

coloboma thus obtained gives a more extensive and peripheral view of the secondary cataract than can be had in cases where the simple operation has been performed. By way of contrast to them, Figs. 96 and 97 represent the secondary cataract in each eye in a case of simple extraction. The pupils have been dilated to give as extensive a view as practicable of the thickened capsule. In both eyes the central pupillary space has remained clear, although there was only the laceration of the capsule obtained at the operation, no secondary capsulotomy having been found necessary. In the left eye there is considerable thickening of the cut edges of the capsule, and the irregular dilatation of the pupil shows that in many places the iris is adherent to it. In the right eye there is a less degree of thickening of the edges of the wound in the capsule, the pupil is round, and there are no adhesions to the iris.

## ON THE CHANGES OF SENILE LENSES WHICH LEAD TO AN INCREASE OF THEIR REFRACTIVE POWER AND TO A DIMINUTION OF THE PRESBYOPIA, AND ON THOSE IN CATARACTOUS LENSES WHICH AT TIMES LEAD TO AN IMPROVEMENT IN THE ACUITY OF VISION, BOTH OF WHICH ARE INCLUDED IN WHAT IS POPULARLY CALLED "SECOND SIGHT."

These changes are of two varieties, — those which occur during the development of the cataract, and those regressive changes which cause a

breaking down or more or less complete absorption of the previously opaque lens-fibres.

I. At some stage in all forms of incipient cataract the lens swells, and as a consequence of this swelling there is a narrowing of the anterior chamber. This is so well known that many practitioners make the subsidence of the swelling which accompanies further degeneration a test as to whether the cataract is sufficiently mature for operation. In consequence of this increase in the curvature of the lens the far point of distinct vision approaches the eye and we have a myopia of greater or less degree. Perhaps also, as Arlt pointed out and as has been lately insisted on by Roure, in cases of nuclear cataract the rays of light used in forming the retinal image come mostly through the periphery of the lens, and are focused nearer to it, thus aiding in the production of short sight. If this intumescence took place in a lens which remained transparent the patient would become less presbyopic, seeing near objects more distinctly without the aid of the glass, while distant objects would be dimmed in proportion to the amount of myopia thus produced. In the majority of instances, however, these changes in the lens are accompanied by so much clouding of its substance that we are unable to measure the exact amount of myopia thus produced, while the outlines of all objects, even when held at the proper focal distance, are so indistinct that the patient derives no benefit even for near work by the change in his refraction. In some rare cases, however, the lenticular swelling is accompanied by so little clouding that a patient who has been previously slightly hypermetropic or emmetropic can now wear with profit and pleasure concave glasses for distance, while for near work he can either entirely lay aside his presbyopic glasses or employ much weaker ones, and he is then popularly said to have acquired second sight. However useful and pleasant this may be to him, nevertheless such swelling of the lens is always accompanied by sufficient clouding to reduce to some extent the acuity of vision, and, although such a patient will often read in a strong light ordinary print fluently, his eyes will usually tire from prolonged use either with or without the aid of a magnifying-glass. We may by careful correction of his ametropia, including the lenticular and corneal astigmatism, in good diffuse daylight have a vision of 6/12 or even slightly better when by increasing the light we make the image more brilliant and at the same time sharpen it by causing the pupil to contract. In such cases we find a large yellow nucleus with a slight but distinct perinuclear haze, while there are no equatorial changes sufficiently developed to encroach on the pupillary area. In fact, careful examination with a dilated pupil and a magnifying-glass behind the mirror or by the use of oblique light shows us layers of opacity less dense than we find them in the zonular cataract of infancy but similarly situated. In one case whose history is given below I could demonstrate a double zone of opacity in an eye which I had often previously examined and knew to have had a perfectly transparent lens. Such processes rarely go on exactly simultaneously in

both eyes, and as a consequence we have the production of considerable anisometropia, the correction of which will often enable us to obtain satisfactory binocular vision. Such cases are not only rare in themselves, but from their very nature seldom present themselves to the ophthalmic surgeon, as they occur among people who are satisfied with the amount of vision which they possess and who, even if the sight eventually becomes very dim, prefer to use what they have rather than submit to the risks of an operation. Owing to the interest attaching to such cases and their comparative infrequency, the salient points in the history and symptoms of three such cases are herewith given.

Case I.—Dr. S., aged sixty-five, formerly a distinguished practitioner of medicine, who states that about his forty-fifth year he put on glasses to read with, and had the year previous to his examination used for near work + 1/15, which he subsequently exchanged for + 1/18. He now finds that for ordinary print he is much more comfortable without a glass, holding his book at sixteen inches, and in this manner he uses his eyes for several hours daily. Within the last six months he has noticed that his distant vision is not so good as previously, and that he gets double images of the moon. O. D. with — 1/16 V. = 20/XL, but everything is a little hazy. There is a slight astigmatism, but the cloudiness of the lens is sufficient to make his answers contradictory. He sees little difference between the vision produced by a spherical glass (— 1/16) and that given by — 1/24 combined with — 1/36 Cy. or — 1/36 Sp. combined with — 1/36 Cy., although the sphero-cylindrical combination makes the lines on the dial more alike. The pupil was dilated and the eye carefully examined with + 16 D. behind the mirror, and also with oblique light. The nucleus of the lens was not as sharply outlined as is usually the case at his time of life; there was perinuclear haze with very fine minute dots, but there were no peripheral striæ. The fundus was healthy and did not present any evidences of myopic changes near the disk. O. S. had a corneal macula dating from an attack of phlyctenular keratitis in childhood, and had not been of much use to him. I never had a subsequent opportunity of examining the eye with dilated pupil, but saw him frequently, and for years his vision remained about the same, and up to his death, in his seventy-fifth year, he was still able to read coarse print with moderate comfort without a glass.

Case II.—*A case where a slightly hypermetropic eye developed in the sixty-second to the sixty-third year a myopia of 3.50 D., and for a time read well without a glass and wore the distance correction out-of-doors with great satisfaction, but where the vision subsequently clouded.*

Mrs. H. D. L., aged forty-nine, complains of headaches and that her reading-glasses are no longer comfortable. She is wearing + 1/20 for near work, and with these glasses can spell out Jr. I with great difficulty at sixteen inches. With either eye she guesses 20/XX, the image of the right eye being the clearer. The pupils were dilated with atropia, and under its use vision sank to 20/L in each eye. With proper correction it again rose to

20/XX. O. D. + 1/60 $\bigcirc$ + 1/60 Cy. axis 105°, V. = 20/XX. O. S. + 1/36 Cy. axis at 40°, V. = 20/XX. The full correction was ordered for habitual wear, and the headache soon disappeared. At the same time she was ordered for near work O. D. + 1/15 $\bigcirc$ + 1/60 Cy. axis 105°, and O. S. + 1/20 $\bigcirc$ + 1/36 Cy. axis 40°. A year later Mrs. L. had become more presbyopic, and was ordered O. D. + 1/10 $\bigcirc$ + 1/60 Cy. axis 75°, O. S. + 1/12 $\bigcirc$ + 1/42 Cy. axis 40°. She used these glasses with comfort for upward of four years, returning again in the autumn of 1880 for other glasses. The eyes were now found to have lost their astigmatism and to have become more presbyopic: O. D. + 1/36 Sp. V. = 20/XX, O. S. + 1/36 Sp. V. = 20/XX, and for reading for each eye + 1/9. Seven years later (June 19, 1888) she again came to be fitted for glasses, and the right eye was found to have become myopic, while the left retained the same grade of hypermetropia. O. D. — 1/48 V. = 20/XXX, O. S. + 1/36 V. = 20/XX. Ordered for distance O. D. — 1/48, O. S. + 1/36, and for reading O. D. + 1/12 and O. S. + 1/9. April 20, 1890, O. D. has become more myopic and the acuity of vision is less. Without a glass V. = 20/C, and + 16 D. behind the mirror shows in O. D. a *slight perinuclear haze.* A year later (April 21, 1891) O. D. V. = 20/CC, and without a glass deciphers with difficulty 0.50 D. print at twelve inches. With — 1/12 V. = 20/XXX only, and cylinders do not improve it. O. S. V. = 20/XXX, and with + 1/12 reads 0.50 at twelve inches. Ordered for distance O. D. — 1/12, O. S. — 1/72 Cy. axis 50°, and for reading O. D. plane glass, O. S. + 1/9. In October, 1892, the vision of the right eye has become more impaired; although she can see the same letters with it, there is a fog about them. When the pupil is dilated with homatropine the subcapsular portions of the lens appear clear, while there is a whitish-yellow, perinuclear, evenly diffused haze. With + 16 D. and transmitted light this zone is clearly seen, and by looking in obliquely a fainter zone of haze is also distinguishable in the periphery to the outside of the inner layers of opaque lens-matter. With — 1/12 O. D. V. = 20/C. Without glass she reads with it 1.25 type at nine inches. There is a long, broad shoot of opacity in the anterior cortical running from the lower outer quadrant upward and inward towards the antero-posterior axis of the lens; but, as it lies mostly behind the iris, it does not materially affect vision. O. S. vision remains 20/XXX without a glass. In April, 1894, the vision in O. D. had become so clouded that with — 1/9 she could see 20/CC only with difficulty; — 1/10 and — 1/12 gave about the same results, and the cloudiness of this eye was so uncomfortable, spreading a fog over the binocular field of vision, that the left eye only was used for reading. The marked failure of vision in O. D. is caused not by any considerable thickening of the perinuclear opacities, but by the formation of opacities in the anterior cortical, there being, in addition to the opaque shoot in the lower outer quadrant already noted as existent in 1892, four ovoid opaque spots lying in the pupillary space and consequently causing the great additional failure of sight.

CASE III.—*A case of second sight in an eye previously emmetropic where, at the age of seventy-five, myopia of 3 D. is developed, which in six years increases to 6 D., while the acuity of vision with correction sinks from 5/20 to 5/40, owing to the increasing cloudiness of the lens.*

Mrs. M. O. D. was first seen by me in July, 1888. She was then seventy-five years of age. As a young woman she had excellent distant vision, and put on convex spectacles for reading at the age of forty-five, and continued to use convex glasses for near work with comfort for a period of twenty eight years. About two years since she found that she could thread a needle without a glass. There is an arcus senilis around the entire cornea, the pupil reacts promptly to light, and there is a strong reflex from the nucleus of the lens. The right eye with — 3.25 has a vision of 5/20, the left eye with — 4 D. 5/20. Using both eyes without a glass she can read Jr. VII. at thirteen inches, but prefers to read with magnifying-glasses, although with the right eye employing + 2 D. and the left eye + 1.25 D. she holds her book by preference at eleven inches, and can read only very slightly finer print (Jr. VI.). The ophthalmoscope shows perinuclear haze in each lens, but no striæ. In the right eye the fundus is best seen with — 3.25 D. The disk is healthy, with slight central excavation and a narrow, semilunar strip of pigment absorption at its outer side, while the red vessels of the chorioid stroma and its black interspaces show over most of the eye-ground, owing to the want of pigment in the retinal epithelium. In the left eye the disk is also healthy, and the absorption of pigment in the epithelium is much less marked than in the fellow-eye. The patient was again seen in 1894, in her eighty-third year. She was still in fair health, the hair of the head had become silver-white, the eyebrows and eyelashes mixed black and gray, the black and gray cilia being nearly equal in number and the gray predominating towards the centre of the lids. She has become more near-sighted, but her acuity of vision, with a proper correcting-glass, has diminished. She can still thread a No. 9 needle without glasses, and can also with the unaided eyes read 1 D. print, and this with either eye alone at six and one half inches. Right eye — 5.50 D. V. = 5/35; left eye — 6 D. V. = 5/40. The ophthalmoscope shows a marked perinuclear haze in each eye, the equator of the nucleus being well outlined, but there are no striæ, either at this point or in the equator of the cortical. By looking obliquely through the pupil another faint circle of haze can be seen, concentric with the nucleus and about half-way between it and the equator of the lens. In each eye, also, there are a few irregular opacities in the anterior cortical, in the pupillary space, and just under the capsule. The eye-ground can still be seen through a decided haze, but the nerves and the absorption of pigment in the retinal epithelium appear about as at the examination six years previous.

Such lenticular changes have received but scant consideration in most works on diseases of the eyes, while many speak of the development of

myopia during the formation of cataract, but little is said of the rare cases where the lens remains sufficiently transparent to enable the patient to pursue ordinary occupations. Ware and Sichel both allude to them, while White Cooper[1] discusses cases "which, having been presbyopic, recover natural sight or become near-sighted." He attributes these changes to an increase in the density and refractive power of the lens, and finds "the sight always a little impaired, and such changes are premonitory of hard cataract." In the last four cases which he has seen and studied carefully at intervals of two or three months he has "traced the change of structure from the first faint indications to the unmistakable characteristics of hard lenticular cataract." Critchett,[2] in a discussion on the development of myopia in the formation of cataract at the Heidelberg Congress in 1873, remarks that we can often restore almost complete acuity of vision by a concave glass. Foerster[3] holds that increase of refraction in commencing cataract is not an exception but very common. Adolph Weber[4] gives two well-observed cases. No. 1, a woman of sixty, with complete cataract in the right eye, and the left, which when first examined had a hypermetropia of 1/40, with a vision of 12/L, had finally a myopia of 1/5, while the ophthalmoscope showed an absence of all myopic changes in the fundus. No. 2, a man who had previously been a good rifle-shot but had no accurate determination of his vision, acquired a myopia = 1/11, which rose under observation to 1/7, and a proper concave glass gave V = 12/L. At the same meeting Meyer[5] remarked that he considered the use of concave glasses in such cases injurious. Nicati[6] had a patient aged seventy-eight complaining of short sight. In the lens there were faint opacities in the anterior cortical "running from the equator to the anterior pole." Two years later these had disappeared, while myopia = 9 D. had developed "with full acuity of vision." In 1877 S. M. Burnett[7] gives an interesting case of second sight. The patient commenced to wear presbyopic glasses at forty, and had several times increased their strength. At the age of seventy he was able to read without glasses. At eighty-five he could read Jr. I. without glass at eighteen inches. V. = 20/L, and with — 1 D. V. = 20/XX. Dr. B. informs me that "the patient died in his ninety-fifth year without the formation of complete cataract." In this case Burnett considered the myopia probably to have been axile. Fuchs[8] relates three cases, in one of which an eye originally emmetropic acquired a lenticular myopia of 3 D.; a second with original myopia = 1.25 D. increasing to 9 D. in

[1] White Cooper, Ass'n Med. Journal, 1853, pp. 996, 997; also in 2d edition of his book on "Near Sight."

[2] Critchett, Klinische Monatsblätter für Augenheilkunde, 1873, S. 461.

[3] Foerster, ibidem, p. 467

[4] Weber, ibidem, p. 463.

[5] Meyer, ibidem, p. 465.

[6] Nicati, Nagel, Jahresbericht, S. 312.

[7] Burnett, Amer. Journ. Med. Sciences, April, 1877.

[8] Fuchs, Klinische Monatsblätter für Augenheilkunde, 1884, S 19.

the right eye, while in the left the myopia increased from 1 D. to 5.50 D.; a third, having been emmetropic, acquired myopia = 6 D. Landesberg[1] gives five cases where hypermetropes with good vision developed a lenticular myopia varying from 1/10 to 1/4½, and claims that in such cases the lens usually presents peripheral shoots with clear pupillary space. Rydel[2] published a case where an emmetrope of seventy-two years acquired in three months a myopia of 3 D., with V. = 20/50, while in eight months the myopia had increased to 4 D., the acuity of vision with correction remaining the same. Rydel[1] describes the nucleus of the lens, as seen with oblique light, as yellow, and its surface "as if fissured," but denies any swelling or increase in the thickness of the nucleus. It appears, therefore, that such senile lenses, while remaining sufficiently transparent to permit useful vision, may develop a myopia varying from 1 D. to 6 D., or even more, while they may for a period of several years remain in this state before the cataractous changes are sufficient to prevent reading ordinary print.

II. In the second class of cases the lens, having been cataractous, clears up sufficiently to permit useful vision. In most such cases there has been a well-developed hard cataract in which softening of the cortical layers has taken place (cataracta Morgagniana) with eventual absorption of the softened matter, while the shrunken nucleus either falls to the bottom of the capsular sac, or, in some less favorable cases, where liquefaction and absorption go on *pari passu*, may remain adherent to the anterior and posterior capsules in the axis of vision, the latter class of cases permitting useful vision only when the pupil is dilated by a mydriatic or when a peripheral iridectomy is performed.

Of late years the number of recorded cases of absorption of cataractous lenses within an intact capsule has rapidly increased. In Otto Becker's masterly and exhaustive essay on the "Pathology and Therapy of the Crystalline Lens" he gives only five such cases,—two by Holscher, where in children with congenital cataract absorption in the intact capsule took place, commencing at the periphery and progressing in the various sectors at an unequal rate, until at the age of five years the pupils were clear; two observed by Warnatz, one in a man aged forty, where both eyes became cataractous and cleared up, the second occurring in a woman of forty-five, where, after lasting five years, the cataract of both eyes acquired a "torn, divided, star-shaped appearance similar to that after keratonyxis." The last of the five is related by Brettauer, and was seen also by Becker himself. The right eye had been successfully operated by Brettauer in 1862, although with considerable loss of vitreous. In 1871 the patient returned for other glasses, and it was observed that the unoperated eye, although possessing prompt pupillary reaction, had a wabbling and greenish discolored iris. After dilatation of the pupil a membrane was seen occupying the pupillary

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[1] Landesberg, Centralblatt für prakt. Augenheilkunde, 1884, S. 101.
[2] Rydel, Klinische Monatsblätter für Augenheilkunde, 1879, S. 92.

space with an adhering radiating gelatiniform mass, the direction of the radii corresponding with the direction of the sectors of the lens. Adhering to these gelatiniform stripes were masses of minute cholesterine crystals. Between these opaque radii red light was obtained from the fundus by ophthalmoscopic examination, while opacities were visible in the anterior portion of the vitreous. With + 1/6 V.= 20/XL with difficulty. The earliest case which I have been able to find in English literature dates to 1858, when Solomon[1] records a case of double congenital cataract where vision was restored by a rapid intracapsular absorption of the lenses. Since 1875 quite a large number of cases are reported in current literature. Szili[2] in 1884 publishes a case where absorption of an over-ripe cataractous lens occurred after an acute attack of glaucoma, the shrunken nucleus remaining and floating with the motions of the eye. With + 11 D. V.= 5/9. Szili inclines to class the case as spontaneous rupture of the capsule and partial absorption of the lens. In the same year Lange[3] reports an interesting case where the cataract was four years in formation, remained ripe for three years, and then commenced to absorb. With + 6 D. V.= 4/60 and with + 12 D. could read Schweigger's 2.25 print. By dilating the pupil with atropia the edge of the lens was everywhere visible, and absorption was evidently going on within the capsule. In the same year Nordmann[4] describes a case of Morgagnian cataract with almost complete absorption of the nucleus. With + 10 D. V. = 6/30. There were minute chalky dots on the anterior capsule and a five-millimetre nucleus, which, falling to the bottom of the capsular sac, prevented correspondingly good vision for near work. At the Heidelberg Congress of 1885 Brettauer[5] reported two additional cases. In one when first seen there was a ripe cataract on one side and an unripe one on the other. The mature cataract was successfully extracted. Fourteen years later the other eye showed an intact anterior capsule, with a shrunken nucleus and cholesterine crystals within it. The fundus was visible with the ophthalmoscope and with + 1/3½ V. = 6/36. The patient was again seen four years afterwards. The remnant of the nucleus had disappeared, and there was a hole at the lower part of the pupil where the capsule appeared to have separated from the zonula. Another case when first seen had a successful extraction in the right eye and a nearly ripe cataract in the left. Thirteen years later there was almost complete absorption of the lens, and the nucleus had shrunk to three millimetres. With + 1/3½ V. = 6/9, rather sharper than in the operated eye. In the discussion Berlin[6] described a case where cataract formed after an attack of iritis, and in a few months cleared up so much that Jr. I. could be deciphered with a strong

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[1] Solomon, Manchester Med. Times and Gazette, February 20, 1858, p. 196.
[2] Szili, Centralblatt für prakt. Augenheilkunde, January, 1884, S. 317.
[3] Lange, Archiv für Ophthalmologie, xxx., 3, S. 211.
[4] Nordmann, Archiv für Augenheilkunde, xiv. S. 165; American edition, 1885, p. 257.
[5] Brettauer, Bericht der Ophthalmologischen Gesellschaft, Heidelberg, 1885, S. 47.
[6] Berlin, ibidem, 1885, p. 53.

magnifying-glass, while Becker[1] spoke of the "spontaneous absorption of a cataractous lens without capsular cataract." In a case of cataracta accreta matura, after specific iritis, absorption commenced within five months after the maturity of the cataract and with + 10 D. V. = 6/36. There had been a successful iridectomy previous to the development of the cataract. In 1886 Lange[2] records a case of Morgagnian cataract where two years later there was complete absorption of the fluid portion of the cataract. In 1887 Kipp[3] describes a case where absorption took place, giving "excellent vision," and in the same year Paul Meyer[4] reports a case out of Leber's clinic where absorption took place after an attack of glaucoma, the inflammation lasting fourteen days. In 1889 Desogus[5] gives a similar case. Gad in 1888 reports another like case. In 1891 Czermak[6] details three cases of intracapsular absorption. The first occurred in an amaurotic eye, the second in an eye where the cataract had been nine years forming, and the third where the absorption occurred after an iridectomy for glaucoma. In all three the shrunken nucleus lay at the bottom of the capsular sac. Natanson[7] describes a case of s nking of the nucleus with absorption of the cortical matter, where through the clear part of the pupil + 12 D. gave V.=5/60. Higgens[8] in 1892 reports a case of almost complete intracapsular absorption of both the peripheral parts and of the nucleus of the lens. + 1/3½ gave V. = 6/36. Lang[9] details a similar case where the patient saw well with a correcting glass. Mitvalsky[10] in 1892 published two interesting and carefully observed cases. In the first O. S. had undergone cataract operation fourteen years previously, and now O. D. spontaneously commenced again to have good vision. The lens had been entirely absorbed except the amber-colored shrunken nucleus, which rested on the lower pupillary margin of the iris, measuring only 2.5 millimetres in equatorial diameter and one millimetre in thickness. The operated eye had with + 12 D. a vision of 1/9, while with the same glass the fellow-eye with the absorbed lens had V. = 1/5. Mitvalsky subsequently removed the nucleus in the capsule by incision in the cornea, and found that the capsule was readily movable upon the nucleus, which weighed only 0.01 gramme. In his second case there had been a previous extraction of cataract in O. D., while in O. S., where a five-millimetre nucleus lay at the bottom of the capsular sac, + 12 D. gave 6/12. The empty capsule was stretched taught across the upper part of the pupil and was transparent, except a few

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[1] Becker, ibidem, p. 54.
[2] Lange, Archiv für Ophthalmologie, xxx., 3, S. 211.
[3] Kipp, Amer. Journ. of Ophthalmology, 1887, p. 157.
[4] Meyer, Archiv für Ophthalmologie, xxxiii., 1, S. 159.
[5] Desogus, Nagel, Jahresbericht, 1889, S. 274.
[6] Czermak, Klinische Monatsblätter für Augenheilkunde, April, 1891, S. 126.
[7] Natanson, ibidem, 1891, p. 423.
[8] Higgens, Ophthalmic Review, June, 1892, p. 185.
[9] Lang, Ophth. Soc. U. K., in Ophth. Rev., June, 1892, p. 106.
[10] Mitvalsky, Centralblatt für Augenheilkunde, October, 1892, S. 289-298.

dotted chalky white spots. The eye-ground was normal. Schneidemann[1] also relates a case of spontaneous absorption of cataract where the disk was plainly visible and partially atrophic; + 11 D. gave 12/CC only. In this case, as the author describes the capsule as being at present only at the upper part of the pupil, there had probably been subsequent spontaneous rupture of the capsule. The clinical facts relating to the spontaneous absorption of cataractous lenses within their capsules have, I think, been fairly set forth by the cases already abstracted. Since these have been reported an increasingly large number have been recorded. Lang, Robinson, Gardiner, Schramm, Egbert, v. Hippel, Alt, Czermak, Delbés, Chevallerau, and Baquis have all published instances. Such processes are all chronic in their nature, and usually extend over a number of years. The most important complications which have so far been noted are glaucoma and detachment of the retina.

### SPONTANEOUS RUPTURE OF THE CAPSULE AFTER PARTIAL ABSORPTION OF THE CATARACTOUS LENS.

In some instances after partial absorption of the cataractous lens the capsule ruptures and empties the remnants into the anterior chamber. St. George Peachy[2] in 1861 mentions a case where a cataract of long standing, in what was apparently a myopic eye, had the capsule ruptured; the amber-colored nucleus, which had fallen into the anterior chamber, lay below the line of vision, so that the patient could read newspaper headings without a glass. Del Monte[3] in 1871 reports such a case where, cataract having existed twelve years, inflammation followed and sight was regained, a small body (nucleus?) being in the anterior chamber, while + 1/4½ gave 15/L vision. Arlt[4] in 1881 at the Heidelberg Congress related a case of spontaneous rupture of the anterior capsule of a cataractous lens. The cataract followed detachment of the retina in a myopic eye of a woman aged thirty-four. The amaurotic eyeball was quiet for four years and then suddenly inflamed, and the lower half of the anterior chamber was filled with lens-remnants, which were subsequently evacuated by an incision in the lower part of the cornea. Bitzos[5] reports a case in a patient of sixty years of age where there was absorption of both nucleus and cortical in a cataract of four years' standing with rupture of the capsule upward and inward. Instances in which *traumatic* cataract has formed in apparently intact capsule and subsequently undergone absorption have already been discussed under the proper head.

*Changes similar to those described in the two preceding sections but less in degree at times improve vision sufficiently to be a great solace to the patient,*

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[1] Schneidemann, Amer. Journ. of Ophthalmology, May, 1894, p. 152.
[2] St. George Peachy, Maryland and Virginia Journal, February, 1861.
[3] Del Monte, Nagel, Jahresbericht, 1871, S. 358.
[4] Arlt, Bericht der Ophthalmologischen Gesellschaft, Heidelberg, 1881, S. 130.
[5] Bitzos, Annales d'Oculistique, April, 1897, p. 276.

as is evidenced in the following case, where the subjective sensations caused by the development, ripening, and subsequent partial absorption of cataracts within their capsules are well recorded by an unusually observant patient. Mrs. S. J. H., an intellectual woman, long editor of a successful magazine, in 1862 at the age of seventy-five years first noticed a curious change in her eyesight. In looking at the heavens at night either Jupiter or Venus, instead of looking like disks of light, appeared in the form of a cross a little bowed to one side. In the following six years her eyesight gradually darkened and spectacles were no longer of use to her. She could see coarse print nearly as well without them. All objects appeared as if in a fog, sunlight had a moonlight pallor, and the stars were entirely lost to view. In 1868 she had a severe attack of catarrhal conjunctivitis with great watering of the eyes and "scalding tears." The severe symptoms lasted a week, but several weeks elapsed before they entirely disappeared. The appearance of the planets had changed, the cross had disappeared, and they looked as large as the moon. They had a purple centre with golden spots and a periphery of yellow-gold color with projecting points, while gas-lights now presented a somewhat similar appearance. The sight had so far failed that she could not even write—in attempting to do so the lines of characters made by the pen would run up in a crooked way to the upper right-hand corner of the paper. In February, 1869, a second attack of conjunctivitis set in more severe than the first, and soon after its subsidence the new moon appeared as seven or eight crescents overlapping each other "spoon fashion," while the left part of the field of vision was a broken mass of light. The full moon looked like nine overlapping full moons with a space to the left as if there were room for four more. The whole glistened like silver in sunlight. The purple centre had almost disappeared. The sun she could not bear to look at except when near the horizon, but he then "displayed the same glories as the moon." After this until 1871 she could not decipher any print even with the strongest magnifying-glasses, but in May of that year she found on rising one morning that she saw more light than usual and could recognize the places of pictures on the walls and of the larger pieces of furniture in the room. This encouraged her to seek advice and I then saw her for the first time. With —1/12 in looking at the upper rows of Snellen's letters at twenty feet she could count the rows but could not distinguish the shape of the letters. With the best (right) eye she could read Jr. XIV. at ten inches without a glass; with dilated pupil in the same eye with —1/10 V. = 10/C. In 1873, three years later, she had become less myopic and vision had improved. With —1/30 V. = 20/CC and with + 1/5 she could spell out Jr. V. at four inches. At this time the anterior chambers were rather deep, the pupillary reaction prompt, and there was slight wabbling of the periphery of the iris in sudden motions of the eye. After dilatation of the pupil with atropia a partially shrunken cataract was visible and a nucleus which, although diminished in size, still lay in the visual axis. By looking in obliquely the margins of the lens were visible,

and when she looked downward, as in reading, a small clear space became visible between the lower margin of the lens and the pupillary margin of the dilated pupil, through which she could read coarse print with a cataract glass. The anterior capsule showed numerous faint striations and more marked opacities towards the anterior pole. Transmitted light showed the remnant of a nuclear cataract with striæ in the posterior cortical, while the periphery of the lens, although comparatively clear, showed faint opacities when examined with + 16 D. behind the mirror. When last seen by me in 1876 the vision remained about the same, and the patient, then in her eighty-ninth year, was well content with the very moderate amount of vision which she enjoyed. During these years she had constantly kept her pupil dilated by atropia without conjunctival irritation, increase of tension, or any other disagreeable symptom.

The following case is interesting as showing how an aged patient, where the nucleus had grown to occupy almost the entire lens, leaving only a thin layer of anterior cortical remaining, may without any swelling of the lens or development of myopia have his vision gravely deteriorated by slight cortical opacities and have it appreciably bettered by their partial clearing up. G. W., aged eighty-four, an unusually vigorous man for his years, had been a land surveyor and had all his life enjoyed excellent eyesight. During the last few years both distant and near vision had clouded, and with his right eye he could no longer decipher any print with his glasses (+ 1/7), while with the same glass he could with the left eye still with some difficulty read the newspaper. The pupils were not dilated, but in the right eye oblique light showed mother-of-pearl glitter of the anterior cortical and the ophthalmoscope some coarse, broad, dark radiating lines in the same part of the lens. The left eye exhibited the same appearances but very much less developed. Five years later the cataract in the right eye had become over-ripe and the anterior cortical had partially cleared and been absorbed. With this eye and with + 1/7 and still better with + 1/5 he could again read with difficulty newspaper print in a strong light, while the left eye had clouded so that it was of no use for near work. He died in his ninety-third year, and a short time before his death could with the right eye still decipher the coarser print of newspapers.

## ANTISEPSIS IN CATARACT OPERATIONS.

Antisopsis has contributed incalculably to the advance of modern surgery and has lessened enormously the mortality and unfavorable accidents, enabling us to do with a fair prospect of success operations which would have been too rash and hazardous to be undertaken before the introduction of these methods of treatment. Antisepsis also has given us much better results from cataract operations than were previously attainable. There is, however, no branch of surgery which has been so little benefited by it as the surgery of the eye. This is by no means attributable to reluctance on the part of its practitioners to benefit by the observations and experience

of their fellows in other branches, but owing to the inherent difficulties of the subject and to the fact that at present, after many trials in various ways and with various antiseptics, we have not been able to discover any method which enables us to render the conjunctival sac perfectly aseptic and to keep it so for a reasonable time after operations. The extreme delicacy of the tissues of the eye and their intense reaction to antiseptics or any other irritants prevent our using solutions as strongly charged with germicides as is practicable with other parts of the body, and the communication by the lacrymal ducts between the conjunctival sac and the nasal passages furnishes a door ever open to infection. The most thorough and repeated syringing of the canaliculi and lacrymal sac will not suffice to keep these passages free from germs, although for a considerable time it very much diminishes their numbers. In fact, any less severe operation than that of obliteration of the lacrymal sac fails to do so. The details of the attempts at closure of the canaliculi by galvano-cautery reported from the Haab's clinic are instructive, and we find in one case after repeated applications of the cautery to the canaliculi that discharge could still be pressed out of the sac.[1] Streatfeild[2] in fact long since urged the advisability of obliteration of the lacrymal sac and canaliculi as a preliminary to cataract operations in cases where there is lacrymal obstruction. The experiments at Gayet's clinic on two hundred and fourteen cases of operation show that, after careful washing of the conjunctival sac with 1-6000 solution of mercuric bichloride, cultures in agar-agar with scrapings from it still gave seventy-eight per cent. of successful inoculations, while after washing with saturated boric acid there were eighty-eight per cent. The more minute and careful experiments instituted at Haab's clinic and reported in detail by Hildebrandt and by Bernheimer are also most instructive; they not only show the species of the bacilli most frequently found, but also establish the fact that the most careful washing of the sac by syringing with solution of bichloride 1-5000 and by gently scraping the everted lids with aseptic cotton freshly immersed in the same solutions failed to free the sac entirely of germs. Bernheimer[3] inoculated his own eye with micrococcus prodigiosus, producing an inflammation with slight hemorrhages under the bulbar conjunctiva and with hordeolum of the edge of the lid. He therefore warns us against the evacuation of the lens by means of pressure with the lid, owing to the danger of forcing out infecting germs into the conjunctival sac. Both experimenters agree that the flow of tears mechanically is beneficial by carrying the bacilli out of the sac, and both found that some varieties were unfavorably affected by the lacrymal secretion, while others, as for example the gonococcus and micrococcus prodigiosus, appear to multiply in the lacrymal fluid. Besides the investigators previously mentioned, Sattler, Mayer, Chibret, von Genderen Stort, Wildebrandt, Bach, Morax, Marthen,

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[1] Hildebrandt, Deutschmann, Beiträge zur Augenheilkunde.
[2] Streatfeild, Trans. Ophth. Soc. U. K., 1884, vol. iv., part iii. p. 126.
[3] Bernheimer, Deutschmann, Beiträge zur Augenheilkunde, 1893, Heft viii.

and Franke have all undertaken experiments to ascertain if it be possible to make the conjunctival sac germ-free. Ahlström[1] maintains that the tears have no germicidal properties, but that they act by dilution and by furnishing a pabulum unfavorable to the development of some varieties of germs. The most interesting fact, however, established by the investigations of Hildebrandt and of Bernheimer is that various germs, such as the staphylococcus albus and staphylococcus aureus, may be present in the conjunctival sac in cases of cataract extraction where the healing has been, from a clinical stand-point, entirely normal, while inoculations of the conjunctival secretion would suffice to cause suppurative inflammation in the corneæ of rabbits. Bernheimer tells us that inoculations of the corneæ in rabbits are far more disastrous in feeble and sickly animals, a fact which entirely agrees with our daily clinical experience in men. These facts lead us into an at present comparatively unknown land in pathology and confront us with one of the great problems of the day, namely, why does a given amount of exposure to toxic influences, whether due to germs or to chemical influence, cause serious or fatal disease in one apparently healthy individual and leave another comparatively unharmed? The various antitoxins seem at present to be furnishing one clue to immunity of some individuals. Leber's[2] experiments have made it probable that the various low organisms found in different parts of the animal economy are not of themselves the active toxic agents. He has shown that when large masses of pure cultures of staphylococcus pyogenes aureus have been destroyed by boiling and an ethereal extract made of the residue he obtains a crystalizable principle called phlogosin capable of producing intense inflammation of any tissue with which it has been brought into contact. From this evidence it would look as if these organisms either secreted the poison or else simply found in it a medium in which they throve and multiplied, and acted merely as carriers of toxic material. In any event, whether they are directly poisonous, or only destructive by generating toxic material or by acting as carriers of it, the clinical fact remains that their presence in wounds is in the highest degree deleterious, and that we should strain every nerve to have instruments, hands, and tissues as aseptic as practicable. The question of the possibility of the continuance of these germs in a dormant state in tissues to be waked into renewed life by traumatism or other pathological condition is most interesting in view of those cases of sympathetic ophthalmia occurring long after the injury and without any demonstrable reinfection of the original wound, and of the occasional cases, like those reported by Romiée, where after operation for cataract without iridectomy, during the course of healing, while V. = 1/3, an attack of articular rheumatism set in, followed by acute panophthalmitis.

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[1] Ahlström, Centralblatt für Augenheilkunde, Juli, 1895, S. 194–200.

[2] Leber, Die Entstehung der Entzündung, Leipzig, 1891.

## THE TREATMENT OF CATARACT.

In spite of centuries of trial, we have hitherto failed to cause the clearing up or the absorption of a cataractous lens either by external applications or by inward medication, and we are therefore obliged to resort to mechanical devices to get rid of the opacity and again to obtain a clear pupillary space through which sharp images may be thrown on the retina when proper lenses are so placed before the eye as to act as substitutes for that which we have destroyed or taken away.

**Reclination and Depression of Cataract.**—The earliest and most widely disseminated method of operation was that by pushing the opacity away from the pupillary space, and some form of what we now know as depression or reclination of cataract has existed among many nations from the earliest times. We have records of this practice in the Ayurvedas of the East Indians, in the works of the later Greek physicians, in those of the Romans, and by the writers of the middle ages. In fact, it was the prevalent method throughout Europe until long after the time of Daviel. The instrument which was used was inserted into the eye either through the cornea or through the sclerotic just behind the corneo-scleral junction, and the lens either dislocated directly downward into the vitreous or turned so that it should rest with its anterior surface looking either directly upward or upward and inward. When such an operation is adroitly performed on a ripe cataract it is sometimes forced out of its capsule, sometimes dislocated with the capsule into the vitreous. In the latter case the pupil is filled by a bead of vitreous, and appears clear and black, and the patient, with a proper lens, is at once able to see distinctly. Where the capsule ruptures, more or less cortical remains sticking to it and occupies the pupillary space, preventing clear vision and becoming denser and more opaque under the action of the aqueous. Sometimes it is gradually absorbed, or else the capsule closes on it and it remains as a permanent opacity in the pupillary space, requiring a repetition of the operation. Where the cataract is soft, it often breaks up under the needle, and large portions of it fall into the anterior chamber; others remain in the pupil and in the posterior chamber, and at times, by their swelling and pressure on the iris, cause effusion of lymph and a formation of synechiæ, or, where the swelling is still greater, glaucoma. Where the operation has been successful, and the entire lens, with its capsule, has been dislocated into the vitreous, it often excites cyclitis and inflammation of the chorioid, and proves also a fruitful source of sympathetic inflammation of the fellow-eye. Mooren says that he has in two years seen twenty-one cases of absolute blindness caused by reclination, and of these cases six had lost the second eye by sympathetic irido-chorioiditis. In some cases, also, it is impossible to get the opaque lens to stay quiet where it has been put, but it rises once more into the pupillary space, or at times falls into the anterior chamber. Since the use of modern methods of extraction, the operation of reclination has fallen

almost entirely into desuetude, a'though a few surgeons still retain it as a method of getting rid of some forms of secondary capsular cataract.

**Discission of Cataract.**—Although every operator who had habitually practised couching or depression of cataract was necessarily familiar with the absorption of flocculi and masses of lens-matter by the aqueous humor, nevertheless the attacking of cataract by discission alone does not seem to have been elevated into a distinct method of operation until Percival Pott, in 1787, extensively practised it. In discission, the capsule is punctured, and slit either through the cornea or through the sclerotica, and the lens more or less extensively broken up by the movements of the needle or knife needle employed. The aqueous thus admitted to the lens causes it to swell and eventually to be absorbed. The main difficulties are the length of time required for complete absorption (a period which usually extends over several months) and the resistance of the hard nucleus of senile cataract. Even these hard lenses have, however, been successfully treated by this method, and some fifty years since it was the prevailing method at the Wills Hospital, then the only eye-hospital in Philadelphia. Dr. Isaac Hays invented a knife-needle for this purpose, and by means of it, introduced through the posterior chamber, the lens was gradually sliced away and cut up. Excellent vision was sometimes obtained, and the author has seen patients thus operated upon who were years afterwards in possession of good eyesight. While by no means advocating this procedure, believing it to be much inferior in its results to properly-executed extraction, it is here recorded as a matter of history, to show that it has been extensively used and that it is practicable even in senile cataracts when undertaken with a sharp knife-needle. One of the most interesting of the annoying sequelæ of such operations of discission on senile cataracts is the frequency of nausea and vomiting within a few hours after, due apparently to pressure of the swollen lens on the ciliary body. Discission by the introduction of a needle or knife-needle through the sclerotic into the posterior chamber is now usually abandoned, most operators preferring the incision in the cornea, attacking the anterior capsule from this point with either a single needle or by the simultaneous employment of two needles whose points are made to cut in opposite directions. Performed in this way, discission has become the ordinary operation for all congenital and infantile cataracts, and indeed for all soft cataracts, as well as for many of the secondary capsular cataracts, and is occasionally used as a means of ripening cataract. In operating by discission it is well to remember that we cannot always calculate on the amount of retraction of the capsule and of swelling of the lens caused by any incision of given size and depth, and also it is extremely difficult rightly to estimate the extent of the wound inflicted by the instrument used for the operation. It is better, therefore, to make our first incision very moderate in size, preferring to wait and see the amount of effect produced, and remembering that it is always possible to repeat the procedure. Indeed, in

most instances we are forced to repeat the operation several times before obtaining a clear pupil, and with absolute asepsis and care in the extent of the incision and the amount of drag which we make on the zonula the dangers of the operation are trifling. In many instances in infantile cataract the anterior capsule is tough and resisting, while the zonule is relaxed, and the lens consequently pushes before the needle and evades to a greater or less extent every attempt to cut it. In such instances the author has found great advantage from the use of the double needle, inserting by delicate rotary motion one fine needle into the lens in order to fix it, and using a sharp knife-needle to cut. In case of the secondary cataracts which form after the operation of extraction, the same procedure not only avoids any drag on the ciliary processes through the zonula, but also enables us better to regulate the size, shape, and position of the opening which we make in the capsule. In operating on zonular cataract we sometimes see that a large incision in the capsule causes the entire nucleus to fall into the anterior chamber, a circumstance always to be viewed with apprehension, but which, however, in the young, owing to the soft and yielding sclera, the natural patulency of the lymph-channels of the periphery of the anterior chamber, and the less liability of the iris to inflammatory reaction, will often pass off without harm by the gradual absorption of the lens-remnants. When, however, after discission sufficient cortical passes into the anterior chamber to interfere materially with the filtration at the periphery of the chamber, the intra-ocular pressure rises to a dangerously high degree, and we have a state of glaucoma. Where this will not yield in a few hours to ice-compresses and rest, it becomes necessary to tap the anterior chamber and let out some of the cortical masses. A moderate puncture, about three millimetres from the corneal periphery, with a lance-knife or a broad needle will usually suffice. In graver cases an iridectomy should be performed. Where, owing to pressure on the iris, a low degree of inflammation is set up, the absorption of lens-matter is either materially diminished or comes to a stand-still, and we must do our best to combat it by the energetic use of atropia, by the abstraction of blood from the temple, and by free purgation. No one who has not repeatedly observed the facts clinically would be prepared to believe the stimulus which is often given to absorption from the anterior chamber by the free use of watery cathartics, always, of course, taking care to proportion their use to the strength and nutritive vigor of the patient in whom they are used.

**Extraction of Cataract.**—Since Daviel introduced the operation of extraction it has gradually gained ground among eye surgeons until at the present time some form of extraction is the method adopted in almost all cases of senile cataract. At first a large flap was universal, but as experience showed that there was everywhere a large percentage of unfavorable results due to bursting of the wound, to prolapse or impalement of the iris, to cyclitis, or to suppuration of the cornea, and as it was supposed that these evil results were largely due to impaired nutrition of the flap, there

was a general tendency developed to extract by means of a meridional or so-called linear wound. This had been employed originally by Gibson and others for traumatic and soft cataracts, and was adopted with modifications by Critchett and by von Graefe for hard ones. To favor the exit of the lens and to place the wound in tissue part of which, at least, had some vascular supply, the wound was made more peripheral, and as owing to its meridional direction and the extremely minute height of the flap it could gape only to a very slight extent, iridectomy became necessary so as to let the lens escape readily and without violence to the iris and surrounding tissue. Preliminary iridectomy was introduced by Mooren with a view of improving the nutrition of the eyeball in cases of complicate cataract and facilitating the extraction of the lens at a subsequent period. Some operators—*e.g.*, v. Jaeger—preferred to extract with a moderate flap, with an accompanying iridectomy, but in the main the ophthalmic world, guided by the teachings of von Graefe, preferred his peripheric linear section or some modification of it. The marked tendency to cyclitis following an incision placed so far back, the small gaping of the wound, the liability to scrape off large masses of cortical from the nucleus during the delivery of the lens, and the difficulty of subsequent evacuation of these masses, together with a tendency of flaps of capsule or of the cut edges of the iris to heal in the wound, gradually led ophthalmic surgeons to enlarge their flap and place the incision entirely in the cornea, so that to-day cataract extraction is performed almost universally with the flap, varying from two-fifths to one-half of the cornea in extent, either with or without an iridectomy. As to the position and size of the incision, almost all the mechanical possibilities have been exhausted by the various operators. As samples of the extremes may be mentioned the incision in the horizontal meridian by Kuechler, and that in the corneo-scleral junction by Jacobson. We must keep in mind that the diameters of the cornea, measured on its anterior surface, average about twelve millimetres for the horizontal and ten millimetres for the vertical diameter, while the posterior surface is nearly circular, its diameters measuring respectively ten millimetres. It is evident that any incision should be large enough at the inner edge of the cornea to permit the ready passage of the cataractous lens without scraping off much of the cortical in its exit. The so-called "simple operation," or operation without iridectomy, the author regards as the normal operation in cases where there is a ripe and hard lens with little soft cortical, and where the iris is active and healthy, dilating readily under the application of a mydriatic, and where the tension of the eyeball is either normal or slightly diminished. Its advantages are that the least possible injury is done to the eyeball, and where it is successful we have a central and fairly mobile pupil of about normal size or smaller, and thus derive all the advantages of a small diaphragm, allowing only the central pencils of light to enter and form images on the retina. Its cosmetic effect also leaves but little to be desired, as it takes minute inspection to demonstrate the cicatrix of the incision, which is usually

also covered by the upper lid, and all that ordinary observation shows is an increased depth of the anterior chamber and a slight wabbling of the iris, both due to the want of the support of its pupillary margin, caused by the absence of the lens. The disadvantages inherent to this method of operating depend for the most part on the size of the flap, the ease with which for the first few days after the operation any coughing, sneezing, or straining on the part of the patient, or any careless handling on the part of the nurse or doctor, may cause rupture of the wound and prolapse of the iris, or at times even of the hyaloid with loss of vitreous. In fact, in some cases the dressing of the eyes and cleansing of the conjunctival sac from its own secretions suffice to call in action the partly reflex, partly purposive cramp-like closure of the lids, and put a strain on the freshly-closed wound every time an attempt is made to open the palpebral fissure. To diminish so far as possible this tendency it is advantageous, after gently pulling down the lower lid sufficiently to partly open the palpebral fissure, to instil a few drops of a two-per cent. cocaine solution heated to 98° or 100° F. In fact, it is a great advantage to have the boric acid solution used to wash the lids and conjunctival sac of this temperature. In such patients it is a golden rule to be satisfied with opening the fissure of the lids simply by gently pulling down the lower lid, washing the fissure with the warm solution, and repeating the manœuvre until the sac and eyelashes are cleansed from all adhering mucus. Gentle wiping of the fissure of the lids and of the eyelashes with bichloride gauze will often facilitate this process. A good view of the wound when desired may often be obtained by placing the thumb on the eyebrow and slightly elevating it, while the patient is told to look gently downward, thus preventing the accompanying descent of the upper lid. In the first few hours after operation there is apt to be backache, with great discomfort and restlessness on the part of the patient, and this can be materially alleviated by frequent change of position made by a competent nurse alternately raising the patient to a sitting posture on a proper bed chair, altering the inclination of the chair, or allowing him to lie flat again on the bed. As to the proper dressing after operation, there is the greatest diversity of opinion and practice among ophthalmic surgeons. It is, I think, evident that theoretically there can be no better bandage than that afforded by the upper lid of the operated eye if it be held gently and persistently in place by the patient. Any addition to this natural bandage heats the conjunctival sac and increases its secretions and favors the multiplication of the microbes which inhabit it, and some surgeons, especially Gayet in Lyons, have treated numerous cases without any bandage, while others use simply a darkened mask to prevent any accidental pressure on the eye. With relays of perfect nurses and with patients absolutely self-possessed and who never forget themselves, there can be no more perfect bandage. Patients endowed with such self-control and quietude are rare, and every surgeon of any experience has only too often seen the various deleterious and often disastrous accidents which may arise from bursting open of the

wound before it is firmly healed. Experience also shows us that a light support to the orbicularis is a great help to the patient in keeping the eyelids closed and immobile, and the author therefore always puts on a light bandage, and is in the habit of closing both eyes, because when one eye is open and is moved the other eye, even under the closed lid, makes a corresponding excursion. Chisholm, however, reports favorable results from leaving one eye free. Hjort has also published the results of the open-wound treatment of cataract, and considers the free escape of the contents of the conjunctival sac caused by the motion of the lids a safeguard against infection. He at first bandaged the eyes for twenty-four hours, but his later cases of one hundred consecutive extractions were treated by the open method throughout. Of these cases treated without bandage he reports six prolapses of the iris, five losses of vitreous, one case of panophthalmitis (considered by the author of hæmatogenous origin), and three cases of such severe inflammation as to leave only light perception or counting of fingers at short distances,—results which do not seem very encouraging. The mental depression and physical unrest which come over most patients in confinement to bed with absolute quietude is to be combated by the constant care of a good nurse, changing the patient's position frequently, without allowing him to use any exertion, and having some one to read to him to help pass the weary hours. If the patient bears confiement well he should be kept in bed until the wound is firmly healed; if not, he should be allowed at any time to be gotten out of bed well wrapped up in blankets and allowed to sit on an easy chair alongside of the bed, thus avoiding the straining incident to putting on ordinary clothing. He should be fed with soft and nutritious food, and in most instances supplied with a moderate amount of alcoholic stimulant. This should never be omitted where the patient has been in the habit of using beer or wine. Where old people become feeble or exhibit any tendency to hypostatic congestion of the lungs they should be promptly gotten out of bed, and quinine, strychnia, and stimulants administered in sufficient doses, while suitable counter-irritation is applied to the chest. The bandage should be removed once in twenty-four hours to cleanse the conjunctival sac of its secretion, and should be discontinued so soon as the wound is firmly healed, as it tends to keep the eye warm and stimulate the conjunctiva to activity, causing it to throw off more mucus and epithelium than it would normally, and affording the best of brood-ovens for the multiplication of any bacilli which may be in the conjunctival sac or may obtain access to it through the lacrymal passages. The light of the room should be moderated by proper shades, and the eyes protected by dark glasses and a frontal shade. In changing the bandage there is always danger of starting the wound if the physician be not exceedingly careful and gentle, and if he does not employ lukewarm solutions to cleanse the eye, but, on the other hand, the removal of the secretion is advantageous, and any germs which may be multiplying are materially diminished in number. Moreover, in many feeble people the eye

may be doing badly without any pain, and it is on this ground also expedient to remove the dressing and examine the eye early. Where there is marked inflammatory reaction iced compresses, aided by the instillation of mydriatics and by abstraction of blood from the temple, have in the author's hands proved the most successful method of moderating and subduing the inflammation. When patients are allowed to go into the fresh air, care should be taken to avoid high wind and dust, both of which are likely to augment any existent low grade of conjunctival inflammation or of ciliary congestion, and to protect the eyes from the glare of light by means of London smoke-glasses. No matter how successful the operation, the use of glasses for near work should not be allowed for two months after the operation, inasmuch as the convergence of the eyes for near work, although the accommodative power has been taken away, is likely to cause ciliary congestion and a thickening of any secondary membrane occupying the pupillary space.

*The Extraction with Iridectomy.*—This is eminently suited to that large class of cases in which there is considerable transparent and semi-opaque and soft cortical matter, because it permits a more thorough evacuation of it, while in the simple operation the manipulations to get rid of it infallibly result in displacing a considerable portion into the posterior chamber behind the iris. It has also the advantage that it can be performed with a slightly smaller flap. On the other hand, the hemorrhage which at times occurs from the iris obscures the field of operation, renders any accurate incision into the capsule difficult, and at times leaves a mass of blood in the anterior chamber after the evacuation of the lens, which under the most favorable circumstances must be absorbed before clear vision can be obtained, and which is likely to leave a delicate fibrinous film adherent to the iris and obstructing more or less of the pupil. Moreover, unless very great care is taken in the toilet of the wound, the edges of the cut iris, uveal pigment, and tags of capsule are apt to become entangled in it and to be the cause of uneven cicatrization and the starting-point of various pathological processes. The amount of light admitted by the coloboma where this is not entirely covered by the upper lid is at times annoying to the patient, and, by admitting peripheral beams as well as central ones, clouds the retinal image. This is often felt in out-door occupations so much as to be a decided disadvantage to the patient, although in the consulting-room with favorable light the vision may be normal in acuity.

In the opinion of the author the combined operation offers better chances of success than the simple operation in most cases where there is much unripe cortical, although he has often had good recoveries from the simple operation in the unripe cataracts of patients in whom the intra-ocular blood-vessel system has been normal and the osmosis and exosmosis active. In these cases, and in all cases of complicate cataract in which the amount of light-perception and state of the tissues of the eye are such as to offer any reasonable prospect of benefit from the operation, the question of *preliminary iridectomy* should be duly considered. In cases with in-

creased intra-ocular pressure and a tendency to glaucoma, and in cases where there are inflammatory adhesions between the iris and the lens, it offers the opportunity of getting the eye in a better condition before we extract the cataract. It has also the advantage over the combined operation that we get rid of all hemorrhage when the lens is extracted, and that we thus obtain a clear field to judge of our capsulotomy. Moreover, like the combined operation, it also enables us better to get rid of any remaining cortical masses. The disadvantage consists mainly in the length of time during which the patient must be retained under observation, and the extraction should not follow the iridectomy in a less period than from four to six weeks, or until all ciliary irritation has disappeared from the eye.

*The Cutting of the Capsule.*—This is probably the most difficult and uncertain part of any cataract operation, for while any sharp instrument carried across it will cause a cut or tear, it is excessively difficult and, in fact, in most instances impossible to make this of any desired shape. The very great diversity of practice among different operators is convincing proof that none of our present methods have arrived at any considerable certainty in the result. It is desirable, if possible, to cut or tear out a central piece of the anterior capsule, and at the same time eminently undesirable to leave a tongue or flap which can be carried into the incision and heal in the wound. A linear incision would appear at first sight to avoid this difficulty, and if it closes promptly to prevent any lens-matter from protruding and coming in contact with the iris, at the same time protecting any remnants of it lying within the capsular sac from free access of the aqueous, and thus avoiding the swelling which so often induces irritation and inflammation of the iris or an attack of glaucoma. It is, however, by no means certain that the capsule will be elastic enough to let out the lens even through a peripheral linear incision into it without its tearing into flaps or tongues which may heal in the wound, although, of course, this is mechanically less likely to happen in the simple than in the combined operation. In any event, by peripheral capsulotomy the anterior capsule will usually be in front of the pupillary space, and owing to the proliferation of its epithelium necessitate a secondary operation, which we are often able to avoid when we succeed in removing a piece of it or in making a large opening into its central portion.

*Extraction in the Capsule.*—Where we are able to remove the entire capsule with the lens we have the most favorable possible conditions for permanently good vision after the operation. In some rare instances in over-ripe cataracts, the lens in its capsule will detach itself from the zonula and the hyaloid, and slip out of the eye with the usual manipulations by which we favor its egress in cases where we have divided the capsule, and occasionally, where there is an over-ripe cataract with thickening of the capsule and a tension rather below normal, we can succeed in removing the lens in the capsule by a sharp hook or wire loop; but in many instances this is impossible. Pagenstecher, who has devoted himself for many years

to the cultivation of this method, and who certainly exercises admirable judgment in his choice of cases, tells us that out of three hundred and fifty-three cases he succeeded in removing the lens in its capsule two hundred and ninety times, while sixty-three times the capsule burst. Hasner has endeavored to secure a clear and axial pupil by puncturing the hyaloid in the fossa patellaris with a needle after the completion of a cataract operation, thus endeavoring to push aside the capsule and secure a clear loop of vitreous in the wound. His example has been followed by some other operators, but the author has no individual experience with the method—a method which in unruly cases or in fluid vitreous would seem to favor a prolapse of vitreous into the wound.

*Prolapse of the Iris.*—In the operation without iridectomy, *if the incision be properly placed*, there will be but a small percentage of immediate prolapse, and when it is simply pushed up against the wound or caught between its lips it may often be reduced by gentle circular rubbing over the cornea on the closed lid. If this does not cause it to retract, or if after a few moments' waiting it shows a tendency to reproduce itself, it is better to perform an iridectomy. More serious are those cases which come from the reopening of the wound within one to three days after the operation. Prolapse, if detected immediately after its occurrence or even on the day following, is best treated by iridectomy, and in nervous patients this should be performed under the influence of an anæsthetic. Where the favorable period has been allowed to slip by, and the iris is partly healed in the wound, it is best not to undertake snipping off of the protruding portions for ten days or two weeks after the operation, and then only where the prolapse appears to increase. As a general rule they call for more complete quiet on the part of the patient and a long persistence in careful bandaging and daily cleansing of the eye. If left to themselves even large prolapses will frequently cicatrize and flatten, but the period of healing is much prolonged, and slight photophobia, lacrymation, and vascular injection along the line of the wound last for weeks. The wound is liable to infection for a longer period, and the distortion of the cornea and consequent astigmatism is much greater. The displacement of the pupil and the drag on the opposite side of the iris are also unfavorable conditions. Even extensive prolapses rarely lead to violent iridocyclitis or to suppuration, but often necessitate a subsequent iridectomy on account of closure of the pupil. Frequently there is no complete prolapse, but some peripheral portion of the iris becomes adherent by its anterior surface to the lips of the wound or partly caught in it, causing the pupil to be drawn up towards this point. Such eyes will often become quiet, with excellent vision. Where in the combined operation the cut edge of the iris remains caught in the wound it should either be gently replaced or seized and cut off. Such impalement cannot be too carefully guarded against, it being a prolific cause of drawing up the pupil with secondary inflammatory processes in the iris and capsule, and in some instances gives rise to cystoid cicatrix, in others to glaucoma.

*Suturing the Lips of the Incision.*—With a view to promote rapid healing of the wound and to prevent prolapse of the iris, sutures have been introduced into the edges of the cut cornea, and, in spite of the additional injury thus inflicted on the cornea and of the opportunity for leucocytes and bacilli to invade it along the course of the threads thus inserted, numerous favorable results are reported. In 1868 H. W. Williams,[1] of Boston, reported forty-four cases treated in this way, and more recently Gayet, Czermak, Suarez de Mendoza, and Kalt have published various cases treated in this manner, apparently without knowledge of Williams's previous work in this direction. Williams employed a fine single thread, and removed it from the fifth to the sixteenth day. Mendoza and Kalt insert their sutures before the corneal incision is made, and bring the knife out between the two points of suture.

**Maturation of Unripe Cataract.**—Following Foerster's suggestion of ripening cataract by massage, large numbers of cases have been treated in this way, some with and some without iridectomy. Opinions vary much as to its value. In the hands of the author, slight frictions on the cornea after opening the anterior chamber not sufficient to cause any reaction on the part of the iris have been most uncertain in their results, rarely producing complete maturation of the cataract. McHardy[2] reports that in nine per cent. of his cases of unripe cataract iridectomy and gentle massage failed to ripen the cataract to the desired point, and that when rougher massage was employed he had seventeen per cent. of violent iritis. Gentle frictions on the capsule itself have been much more effective, but are open to the objection of having to introduce an additional instrument into the eye and to the liability of rupture of the zonula in cases of restless and nervous patients. Fig. 62, at page 318, shows the result of such capsular friction. Ripening of senile cataract according to the plan of Muter, by incision in the anterior capsule, has been rejected by most eye-surgeons on account of the danger of too great swelling of the lens, with consequent inflammatory reaction.

**Getting Rid of Cortical Remnants in the Anterior Chamber and Pupillary Space.**—This is usually best effected by careful stroking of the cornea either with a spoon or by means of the finger on the closed lids. When this fails, careful washing of the lips of the wound by means of gently squirting warm boric acid solution upon them should be resorted to. If necessary, the wound may be made to gape infinitesimally by gentle pressure on the ball, and the anterior chamber washed out by repetition of the same process. The use of even weak solutions of mercuric bichloride for this purpose is followed by opacities of the cornea. The history of washing out the anterior chamber is interesting. St. Yves advocated it to remove inflammatory exudates. Guerin, in 1773, and Sohmer, in 1779,

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[1] Williams, Trans. Amer. Ophthal. Soc., 1866, p. 45, and 1868, p. 58.

[2] McHardy, Edinburgh International Congress, August, 1894.

used it to remove cortical remnants. Both he and Casaamata, in 1782, appear to have used water with alcohol for this purpose. This method of procedure failed to obtain a foothold in practice,—Guerin, Pauli, and Himly mentioning it only to condemn it. It was not renewed until the advent of antiseptic surgery, since which time it has been used in degrees varying from that above described to the introduction of the beak of a syringe into the wound, or complete introduction of a hollow tube into the anterior chamber and washing it out either with distilled water, boric acid, or Panas's solution. The author believes it preferable not to introduce any instrument into the eye for the washing out of cortical remnants.

**Treatment of Secondary Cataract.**—As has been already described in the section on clinical appearances, we often have such folding and thickening of the capsule coming on at varying periods after cataract operations as materially to diminish vision. Where such changes are limited to the anterior and posterior capsules, we usually readily reëstablish good acuity of vision by cutting or tearing the film, and, where this is tougher than usual, being careful to avoid any traction on the zonula or ciliary processes, either by the introduction of a second needle or knife-needle through the same rent in the capsule, when the careful separation of the two instruments will usually secure a good result. When, however, such secondary cataract has been accompanied by plastic iritis, by drawing up of the capsule into the extraction wound, or by the development of glaucoma, we have conditions which tax all the ingenuity of the surgeon, and only too frequently, in spite of his best efforts, lead to blindness. It has been claimed by Roeder that every operation for secondary cataract leads to a change in the curvature of the cornea and a consequent variation in the amount of astigmatism. While in some cases where secondary cataract has been dense and thick the author has found such changes, in the majority of instances the patients will pick out the same cylindrical correction which they did before the secondary operation.

## THE ASTIGMATISM PRODUCED BY CATARACT OPERATIONS.

A certain amount of astigmatism is almost sure to be produced by the cicatrizing process, and it ensues with such regularity that it is often possible by simple inspection of the eye to ascertain within a few degrees the position in which the axis of the convex cylindrical correcting-glass should be placed. This is usually in a position parallel to a line joining the points of puncture and counter puncture, the cicatrization causing a flattening in the meridian at right angles, and in cases where this is considerable there is a slight bulging of the meridian which is parallel to the direction of the incision. While, as just stated, the resultant astigmatism has usually the meridian of least curvature at right angles to the line of incision, an observation of Laqueur shows that it may occasionally be in opposite directions. Such instances are either due to the existence of a high grade of astigmatism before the operation, the meridian of greatest curvature being

at right angles to that chosen for the incision, and the counteracting influence of the operation not being sufficient to counterbalance it, or to some unexplained irregularity in the healing process, the edges of the wound rucking up against each other instead of overlapping as usual. As might be expected, the astigmatism is usually greater when the flap has been large than when it is small, and my record-books show, as a rule, much lower astigmatism after iridectomy and after peripheric linear operations (Graefe's operation) than after flap extractions. It is always greater where a prolapse of the iris has been allowed to cicatrize in the wound, or where a mass of consistent vitreous has come to occupy the same position and remained there during the healing process. It is rare that acuity of vision is not materially increased by a careful cylindrical correction in a sufficient degree to be gratefully appreciated by the patient, and it is singular how long this fact has been overlooked by the bulk of the profession, and how few operators, even to day, carefully correct their cases. Yet I have seen successful operations for cataract where the patients were dissatisfied with the result, and, in spite of clear media, could not read any ordinary print, who have been most agreeably astonished when provided with a proper sphero-cylindrical correction. The amount of astigmatism is usually greater soon after the operation, and it often diminishes so considerably within a few months that a weaker cylinder than that selected four to six weeks after operation will much improve the vision. Donders, in his classic work on refraction, long ago called our attention to the fact that regular astigmatism frequently followed the operations for cataract as well as those of iridectomy, but does not seem to have followed up the subject more minutely. It has of late been the subject of much study and discussion by various authors. From the time of the completion of the incision in a cataract operation, the curvature of the cornea varies with the pressure of the eyelids on the eyeball, and with the action of the muscles of the eye every time that the eyeball is moved under their influence. Baiardi, in examining such eyes with the ophthalmometer, found that each time the eye winked and closed sufficiently to bring the cilia in the field of the instrument the corneal images approached each other in the vertical meridian (extraction having been performed upward), while they separated in the horizontal meridian. Jackson[1] has given us a careful study of the development and course of astigmatism after cataract operations. He says that either simple inspection of the cornea or the use of Placido's disk shows that the form of the cornea remains normal until the anterior chamber is re-established; "then, in the course of two or three days, or sometimes in a few hours, the corneal asymmetry becomes evident and quickly reaches its maximum. From this time onward the tendency is for the astigmatism to diminish, until at length a permanent condition is reached." The same author tells us that in sixty per cent. of the cases

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[1] Jackson, Ophthalmic Review, December, 1893, pp. 349–360.

the change continued between two and three months; that in twenty per cent. change continued in the third month; but that in fifteen permanence was reached in the second month. Dolganoff[1] gives the average astigmatism (as determined by Javal's ophthalmometer) to be 4.9 D. two weeks after the operation, sinking to 2.8 D. three to four weeks after the operation, and to 2.6 D. five to six weeks after. Most writers agree (Chibret, Oswalt, Jackson, and others) that the determination of astigmatism with Javal's ophthalmometer gives higher results than the examination with glasses. It is evident, however, that it is impossible to lay down any definite rule as to the amount of astigmatism occurring after a cataract operation, as this will necessarily vary with the location, the character, and the extent of the incision, with the increased or diminished tension of the eyeball, with the accidents during or after operation (such as loss of vitreous or prolapse of the iris), and with the amount of subsequent inflammation and of effused cicatrizing material, so that each case will be a rule for itself, and we will be obliged to study it carefully with the aid of test-lenses. There have been numerous attempts to remedy the astigmatism produced by operations by incisions made into the cornea to counteract it, so placed as to bring about contraction in the meridian of greatest curvature. Thus, Baiardi, Bates, Faber, Schiötz, Reymond, and Lucciola have given us their experience in such operations. The last author has done ten such operations, and finds the amount of flattening in the meridian of greatest curvature thus produced averages about 1 D. He further states that where the incision does not open the anterior chamber, but only effects a loss of substance in the cornea, there is an augmentation of curvature in the meridian parallel to that in which the incision is practised. More recently Lans (1898) has proved that in a rabbit's cornea non-perforating wounds, two in number, one at each end of a given meridian, placed two millimetres inside of the limbus, and extending one-fourth of the circumference of the cornea and two-thirds of its thickness, produce, after cicatrization, a permanent astigmatism, from 3 D. to 6 D., the most curved meridian always running parallel to the incisions.

## REMOVAL OF THE TRANSPARENT LENS FOR THE CURE OF MYOPIA.

The frequent good results of the displacement or of the extraction of cataract in myopic eyes, and the much weaker glasses that such patients are able to wear, have given many surgeons the idea of correcting high degrees of short sight by the removal of the transparent lens. As long ago as 1708 Boerhaave[2] tells us that he had observed a cataract in a myope where after operation the patient could see well without a convex glass. Desmonceaux[3] in 1776 advised the extraction of the lens in cases of high

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[1] Dolganoff, Archiv für Augenheilkunde, xxix. S. 18.

[2] Boerhaave, Abhandlungen von den Augenkrankheiten, 1708, S. 276.

[3] Desmonceaux, Traité des Maladies des Yeux et des Oreilles, Paris, 1776, t. i. p. 406, and t. ii p. 140.

myopia in the young, and says that he has several times seen the operation practised with success. The idea, however, does not seem to have met with acceptance, and, although we find Richter [1] proposing the depression or the extraction of the lens for the cure of myopia, he does not appear ever to have performed it, while Beer,[2] although referring to Desmonceaux's proposition in his Repertorium, and considering it more fully in his treatise in eye diseases, realized its dangers and difficulties, but thought it would be worth a trial in the event of any high-grade myope being willing to submit at least one eye to this mode of cure. At the Heidelberg Ophthalmological Society in 1858 Adolph Weber stated that he had several times removed the lens with good effect in high myopia, and at the same meeting Mooren also reported a case. The advisability of the operation was most unfavorably discussed by v. Graefe and by Donders, and this, together with an unsuccessful case, seems to have caused Mooren to abandon the operation for a time. Both theory and practice appear to have slumbered until Fukala in 1890 reported five cases in which he had performed the operation, and in 1893 the same author published observations on eyes thus operated which he had watched for several years, and claimed that he had thus made people who were unable to gain their daily bread once more fit to pursue their occupations. At that time he operated only on young people with a high degree of myopia and rejected those with advanced chorioidal changes. As samples of his favorable cases may be quoted: I. A teacher twenty-two years of age with M. = 20 D. After operation the myopia had been reduced to 5.50 D., and the patient read without a glass at his far point and wore full correction for distance. II. A boy of twelve years with M. = 15 D. The operation rendered him nearly emmetropic, and he used + 4 D. for reading. Fukala operated by discission and subsequent evacuation of the crumbling and absorbing lens-masses. About the same date (1890) Vacher in a paper before the French Ophthalmological Society advocated the removal of the clear lens by extraction in high myopia, and reported cases. Since that date the operation seems to have received tolerably wide-spread acceptance, and many cases have been reported by Pflüger, Schweigger, Thier, v. Hippel, Vossius, Sattler, Wicherkiewicz, Schroeder, Laqueur, Widmark, Hoerstmann, Pergens, Eperon, Fergus, Lawson, and others. In his last work on this subject Fukala [3] has collected from various sources one thousand cases, and still more recently at the Moscow Congress quotes two thousand cases as already reported in the literature of the subject. Pflüger in 1893 had already operated on twenty-five such eyes, had examined his patients subsequently, and found that all were satisfied with the results except "one who hurt his eye in delirium tremens," and that there was usually improvement in the acuity of vision. Schweigger, however, in reporting his cases does not agree that there was any betterment in the

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[1] Richter, Anfangsgrunde der Wundarzen·i-kunst, Wien, 1790, Theil iii.

[2] Beer, Lehre der Augenkrankheiten, Wien, 1817, Theil ii. S. 659.

[3] Fukala, Heilüng hoechstgradiger Kurszichtigheit, Wien, 1896, S. 87.

acuity of vision. Fukala and most of those who have found improved acuity of vision attribute it to the larger retinal images and to the greater illumination of the retina obtained by such eyes when they are able to lay aside the high concave glasses previously necessary. Many of those who have thus operated agree that the removal of the lens in cases of myopia varying from 14.5 D. to 16 D. leaves the eye nearly emmetropic, while others maintain that a previous myopia of 20 D. is necessary to this result. The variation in these results is very great, but it is nevertheless a remarkable fact, since we know that in average eyes the operation for cataract alters the refraction of the eye about ten diopters. It is probably due mainly to the great length of axis in eyes with high myopia and in part to the atrophy of the circular fibres of the ciliary muscle which is characteristic of this state of refraction, thus allowing a relaxation of the zonula and an increase in the convexity of the lens, the operation having usually been performed on young subjects where the lens is still elastic and resilient. Moreover, Vossius has called attention to the almost globular shape of the lens in a patient with myopia equal 30 D., while Sattler maintains that the lenses in high myopia do not undergo sclerosis in the same way and to the same degree as those in hypermetropic and emmetropic eyes. A case in which Widmark operated on a man of seventy years of age for the cure of myopia of 9 D. seems to confirm this view, since the removal of the lens diminished the refraction only by this amount, leaving the eye emmetropic. This is certainly the minimum amount so far recorded, while, on the other hand, Vossius, who determined the refraction by the shadow test, the glass being held close to the examined eye, found that in a case of myopia = 30 D. there remained after the operation only 1.5 D., the absence of the lens in this instance causing a diminution of refraction = 28.5 D. Of course the apparent difference in refraction before and after operation will depend very much on the accuracy of the primary determination, and in the use of lenses so powerful as 15 D. to 30 D. a few millimeters in the distance at which the glass is held from the cornea will make a great difference in the result, a much stronger glass being required for each additional millimetre of distance. The testing glass should be held either almost in contact with the cornea, or, better still, a careful subjective determination of the far point or an objective determination of it as recommended by Schweigger should be adopted. This latter method consists of an objective determination of the anterior focus of the eye. A small electric lamp is so held as to throw a brightly illuminated image on the retina, the return rays are caught on a screen, and the distance measured between this screen and the plane of the iris. It is also to be remembered that the number of diopters which any given eye loses in its refraction by the removal of its lens will vary with its length of axis, being greater in an elongated eyeball than in an emmetropic one. Furthermore, even in an eye with an axis of given length we would have a greater optical result from the removal of the lens when the anterior chamber is shallow, and the distance between the principal point

of the lens and the fovea greater, than we would have in a deep anterior chamber, where this distance would be shorter. The patients submitted to the operation have usually been young, but Pflüger reports a successful result at the age of forty-eight, Sattler one at the age of sixty-four, and Widmark (as previously stated) one at seventy years.

Unfortunately for absolute accuracy in the estimation of the probable state of refraction after removal of the lens, we cannot expect the ordinary dioptric formulæ to serve us accurately, as we know only the radius of curvature of the cornea, while the curvatures of the lens, its index of refraction, and the distance of its optical centre from the fovea are all unknown quantities. It may be assumed from the measurements of Donders, confirmed by those more recently made by Otto,[1] that the cornea in high myopia is not more convex than usual. According to Ostwalt,[2] we obtain a rough approximation to the refraction after operation by dividing the number of diopters in the correcting glass before operation by two, and subtracting the value thus obtained from the refractive value of the emmetropic normal eye after removal of its lens. Hirschberg[3] adopts very similar views and gives as examples (taking 10 D. as the value of the aphakic emmetropic eye):

$$\text{M.} = 20\text{ D.} + 10 - 20/2 = 0 \quad = \quad \text{E.}$$
$$\text{M.} = 15\text{ D.} + 10 - 15/2 = 2.50\text{ D. H.}$$
$$\text{M.} = 24\text{ D.} + 10 - 24/2 = 2\text{ D.} \quad \text{M.}$$
$$\text{M.} = 30\text{ D.} + 10 - 30/2 = 5\text{ D.} \quad \text{M.}$$

It is evident that in every instance the result will depend on whether the fovea does or does not lie at the principal focus of the cornea. When it does so lie we have emmetropia; when it falls in front of it we have residual myopia; and when it falls behind it, resultant hypermetropia. The operations adopted to get rid of the transparent lens have usually been discissions, or discissions followed by linear extractions. In considering the possible dangers to the patient after such an operation, there is the small but ever present risk of suppuration in spite of the most conscientious employment of any asepsis or antisepsis at present at our command. The risk of glaucoma coming on soon after the operation seems to be reasonably within our control by the prompt evacuation of any swollen lens-remnants, but there remains the possible awakening of low-grade inflammatory processes and the increased possibility of detachment of the retina, a calamity to which myopic eyes are notably disposed, even when no extraneous influences have been put in motion to cause inflammatory processes. Several such detachments have already been reported (Sattler, Valude, Wecker, Wray, Van Millingen, L. L. Johnson, etc.) as occurring in myopic eyes which have undergone the operation for removal of the lens even where the immediate results have been most satisfactory. Fukala, how-

[1] Otto, Archiv für Ophthalmologie, xliii., 3, S. 547.
[2] Ostwalt, Centralblatt für Augenheilkunde, 1895, S. 388.
[3] Hirschberg, ibidem, 1897, p. 68.

ever, does not seem to think that the tendency to detachment existing in myopic eyes after the removal of the lens is greater than that existing in any eyes with equally high grades of myopia, and cites Mooren's statistics, that of five thousand six hundred and thirty-one cases of myopia, with all possible grades of sclerotico-chorioiditis posterior, there were one thousand two hundred and seventy-three cases with one-sided detachment, or 22.88 per cent., and eleven cases of detachment in both eyes. Prolapse of the vitreous at the second stage of the operation—*i.e.*, when the incision is made in the cornea to evacuate the fragments of the lens—does not seem to have occurred as frequently as might have been anticipated. The statistics showing the results of operations for the removal of clear lenses in myopic eyes may vary greatly. Mooren,[1] who reports one hundred and fifty-six operations on eighty individuals, had none but successful cases. The results published by other expert operators, however, are sufficient to show that it is by no means an operation devoid of risk, and we ought always to remember that these risks should weigh all the more heavily inasmuch as we are operating on seeing and not upon blind eyes. Thus, the Leipzig clinic[2] reports, out of seventy-eight cases, four absolute losses and three badly damaged eyes. Haedicke[3] publishes fifty operations at the University of Berlin Eye Clinic in which there were seven cases (fourteen per cent.) of retinal detachment. Darier[4] gives one hundred and forty-two cases of various operators in which five per cent. of the eyes were lost. The losses were due three times to infection and four times to detachment of the retina. Gelpke[5] gives the statistics of fifty-nine operations on forty-six individuals, and in twenty-two eyes—*i.e.*, in thirty-eight per cent. of the cases—glaucoma developed in the later stages of healing. It usually, however, yielded to the instillation of myotics. Schnabel[6] tells us that he has never seen a case where the *near vision* after operation with the use of a convex glass has been as good as it was before the operation, either with the unaided eye or with a weak concave glass. These results, while they ought not to make us overlook the large number of favorable results reported, should make us weigh carefully the chances of success in any eye which still has a reasonable amount of vision. Most authors seem to assume that where the myopia has reached from 15 D. to 20 D. a correction for distance is not borne by the patient. Such, however, has not been the experience of the author, and although the prismatic distortion of images even in carefully centred concave glasses of such strength is very annoying, and although in many cases vitreous opacity and chorioid

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[1] Mooren, Die medicinische und operative Behandlung Kurzsichtiger Stoerungen, 1897, S. 118–135.

[2] Otto, Archiv für Ophthalmologie, xlii., 3, S. 543.

[3] Haedicke, quoted in Centralblatt für Augenheilkunde, Juli, 1898, S. 214.

[4] Darier, Bericht ueber den Kongress in Moskau, 1897.

[5] Gelpke und Bihler, Beiträge zur Augenheilkunde, 1897, Heft xxviii.

[6] Schnabel, Wiener medizinische Wochenschrift, 1898, Nr. 23.

changes materially reduce the acuity of vision, still many of them find great solace in the use of distant glasses, and make out to do a good deal of near work by using one eye at a time without a glass and holding the objects very near. In many other cases a partial correction gives the patient a moderate but very agreeable accommodation for near work and does away with the disagreeable necessity of holding the work excessively near to the eye. Schweigger is in the habit of operating on one eye for distant vision, and allowing the patient to use the other for near work. I have seen a case which I could follow from the age of sixteen to the age of fifty, with myopia of 20 D. in each eye, accompanied with marked thinning of the chorioid and stretching of the retinal vessels, but without any atrophic conus. The patient at first used —18 D. for distance, and afterwards —20 D., giving V. = 20/LXX, employing the eyes alternately without glasses for reading. At the age of forty-eight, after an attack of gouty iritis, retinal detachment occurred in one eye, but the patient can still read comfortably with the other without a glass, and has with full correction a vision of 20/C. Another patient has worn for years with comfort O. D. —21.50 ⊂ — 2 Cy. axis 135°, O. S. — 20.50 ⊂ — 2 Cy. axis 30°, this correction giving 5/25 in each eye, while with binocular vision the acuity rises to 5/20. For reading O. D. — 18.50 ⊂ — 2 Cy. axis 135°, O. S. — 17.50 ⊂ — 2 Cy. axis 30° enable the eyes to do much useful work. Such high degrees of myopia as make operation desirable would seem to be much more frequent in Europe than with us, and have certainly been great rarities either in the author's private or in his hospital practice. Where he has encountered them, the already existing useful although impaired vision, with the risks of operation, the loss of accommodation, and the danger of exciting any chorioidal or retinal changes into fresh and augmented activity have always kept him from operating.

## SOME OF THE CALAMITIES AND ACCIDENTS WHICH AT TIMES ACCOMPANY OR FOLLOW OPERATIONS FOR CATARACT.

There is probably no serious surgical operation which, granting proper selection of the case, skilful performance, and careful nursing, will give a higher average of success than the operation of extraction of cataract. The portion of the eye involved in the operation is small, the blood-vessel system is but little implicated in any case, and in some forms of operation not at all, and we usually therefore are not prepared to expect grave results to the general health, however bad our prognosis may be as to the restoration of useful vision. Nevertheless, grave constitutional disturbances will often follow the operation, and at times death may result from it. In most fatal cases it is less the direct result of the operation than the impetus given by the shock and confinement to some previously existent pathological process which may at the time be in abeyance, but which once started is powerful enough to carry the patient, whose powers of resistance are usually enfeebled by his age, rapidly down the hill to the valley of death at the bottom. It is

rare to see the inflammation of the eye after an operation become the starting-point of fatal disease, the remarkable cases of Brettauer [1] and of Webster,[2] where meningitis, due apparently to panophthalmitis, carried off the patient, being rare exceptions. In the case of Arlt,[3] where panophthalmitis set in and was followed by pneumonia a month after the operation, the reported details do not show whether the pneumonia was embolic or whether it was simply a consecutive accidental disease. On the other hand, pathological processes sequent upon cataract operations often exhaust the strength and reduce the vitality, rendering the patient a prey to intercurrent disease, and often also are the starting-points of sympathetic irritation and sympathetic cyclitis in the fellow-eye. The physical and mental depression dependent upon confinement and separation from family and accustomed surroundings is often very great, and I have repeatedly seen old people from country districts really ill, losing appetite and failing in strength, from sheer home-sickness, even when their operated eye was recovering without any bad symptoms and with every prospect of useful vision. Of course such mental states are more intense and intractable where for any reason the eye is doing badly and the prospects of useful sight are diminished. The effect of bandaging the eyes or of confinement in a darkened room is at times also a powerful depressant; v. Fraenkel-Hochwart [4] cites a case where bandaging of itself without operation brought on a delirium, which disappeared at once without treatment on removal of the cause. Warlomont has seen it produced by simple closure of the eyelids. The advantages gained by the early removal of the bandage in keeping the patient cheerful and content are often a temptation to remove dressings from nervous patients, and cause us to hesitate and balance these advantages against the increased chances of rupture of the wound and other accidents which only too often follow the injudicious exertions of ignorant or disobedient patients. This form of delirium is quite distinct from that occasionally produced by the instillation of mydriatics either before or after an operation. The same depressing influences render the system a prey to any latent disease.

*Malarial Fever.*—I have several times seen patients from malarial districts who had previously had malarial fever but who had been free from it for years, and who considered themselves in their usual health, develop an attack of intermittent fever within a few days after the operation, and I once saw a patient eighty years of age have a severe attack of gout in the big toe develop on the fifth day after a cataract operation, accompanied by a sharp attack of iritis in the operated eye. Verneuil reports a case of extraction in a patient with chronic gout, aged sixty, where for the first two

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[1] Brettauer, Becker, Graefe und Saemisch, Handbuch, Bd. v S. 381.
[2] Webster, Archives of Ophthalmology, 1888, vol. xvii. pp. 25-28.
[3] Arlt, Becker, Graefe und Saemisch, Handbuch, Bd. v. S. 382.
[4] Deutsches Wochenblatt für Gesundheits-Pflege und Rettungswesen, 1891, Bd. iv. S. 152.

days all went well, but later attacks of gout with headache, delirium, and somnolence set in, followed by death on the twenty-fourth day. Rockcliffe[1] has recorded a case of alarming hæmatemesis after operation, followed by acute gout in the hands and feet on the second day, and by intense chemosis of the conjunctiva on the sixth. The patient, notwithstanding, recovered with a vision of 6/9. In the discussion, Nettleship thought acute gout following cataract operation not uncommon; he had met with it several times; while Lang had seen four cases, all of which had recovered without any ocular manifestations.

*Facial erysipelas* is a serious complication of operations either for cataract or glaucoma, but I have seen a patient recover from a severe attack with a good eye, the disease having developed on the third day after an iridectomy for glaucoma.

Patients with asthma, chronic bronchitis, naso-pharyngeal catarrh, etc., are likely to develop hypostatic *pneumonia* if kept too long in the recumbent position, to say nothing of the dangers of springing of the wound by sneezing and coughing. There are also several recorded cases of fatal pneumonia setting in soon after the operation, apparently without any good cause. I well remember an instance where violent double pneumonia set in in an apparently healthy negro on the fourth day after operation, and where before death the flap, which had seemingly united, became loosened and fell down over the cornea, leaving the wound wide and gaping.

I have also known sudden death to occur in patients with fatty degeneration of the heart and subject to occasional attacks of angina pectoris, but who previous to the operation had been in what for them was a fair state of health, and have been led to consider such states of the system as grave complications of cataract.

Mild wandering delirium without apparent cause will sometimes set in after cataract operations and cause the patient to attempt to get up and walk about, and tug at his bandages and sometimes to succeed in removing them. Such patients forget where they are and have hallucinations, hearing voices of people who are not present talking to them, and in rare instances have violent delirium. The author recollects a case of simple extraction, with the eye doing well, where the patient wanted to throw him out of the window, because he heard his wife (who was over a thousand miles distant) talking in the hallway and he was not permitted to see her. The eye eventually recovered with 20/XX vision. There is no fever with the delirium, and it comes at times in people who have never been in the habit of using alcoholic stimulants. It usually yields promptly to the administration of stimulants and mild doses of opium or chloral. In operation on any patients previously addicted to the use of alcohol, even when not using it to excess, it is important to continue their daily ration, and this is even more the case

[1] Rockcliffe, Trans. Ophth. Soc. U. K., June 11, 1896, p. 352.

in people who have been hard drinkers. Chisholm[1] reports a case of furious delirium in a patient eighty-two years of age after cataract operation.

*Bright's Disease.*—How far Bright's disease of the kidney may be a source of danger in cataract operation my individual experience does not allow me to decide. Many writers are of the opinion that the prognosis of cataract operation as far as the eye is concerned is very bad in such cases. Sichel tells us that the prognosis of cataract operation undertaken on patients with this disease is uniformly unfavorable, and contrasts it with the good results obtained by him in diabetics. The author[2] has elsewhere reported two deaths within a few days after cataract operation, due apparently to transient congestion of an already diseased kidney caused by the administration of ether as an anæsthetic, although every surgical clinic furnishes frequent evidence that such is not necessarily the effect of ether anæsthesia in all cases of Bright's disease.

*Diabetes Mellitus.*—Advanced cases of diabetes mellitus sometimes give fatal results after operation. Thus, Becker reports a case where, after extraction of cataract in one eye and preliminary iridectomy in the other, the patient died of diabetic coma on the second day. The author has also seen a case where after an operation for diabetic cataract the cornea became gangrenous, as did also apparently the brownish and discolored but consistent vitreous, which subsequently protruded from the wound and where death ensued from coma a week after the operation. He also well recalls a case where low-grade iritis with slight hemorrhages in the vitreous followed the operation in the second week, and the patient soon after died from gangrene of the hand and forearm, supervening on an accidental blow against the corner of an iron bedstead.

## SEQUELÆ WHERE THE BAD RESULTS ARE CONFINED TO THE EYES THEMSELVES.

**Panophthalmitis.**—Sloughing of the cornea and suppuration of the eyeball with subsequent shrinking are certainly less common than they were before the days of aseptic surgery, but we have not been able to banish them altogether from the records.

**Plastic cyclitis** is only too frequent, and occasionally gives rise to sympathetic disease of the fellow-eye. The percentage of cases giving rise to sympathetic disease is difficult to determine, as the sympathetic inflammation usually develops after the discharge of the patients, and they often wander to other clinics. Becker reports three cases of sympathetic affection out of twelve hundred and sixteen cataract operations. It is sometimes rapid in its course and sometimes malignant, as in a case of Knapp's, where it broke out on the fifty-sixth day, and where the sympathizing eye was lost in spite of the prompt enucleation of its fellow. Sympathetic in-

[1] Chisholm, Richmond and Louisville Med. Journ., January, 1873.

[2] Norris, Transactions of the Amer. Ophth. Soc., 1881, p. 189.

flammation is supposed to occur more frequently after the combined than after the simple operation.

**Prophylactic Enucleation.**—Such complications bring up the question of prophylactic enucleation, and the author has always been in the habit, where the eye on one side was shrunken and tender to the touch as a result of previous operation or injury, of enucleating this eye before undertaking cataract operation on its fellow. Pechdo[1] records two cases where this precaution was neglected, and the operated eye attacked by plastic cyclitis, which set in in the second week after the operation and entailed the entire loss of the operated eye.

**Hemorrhage: Retrochorioidal and other Forms of Intra-Ocular Hemorrhage.**—Hemorrhage occurring from rupture of the vessels of the chorioid either during the operation or immediately afterwards is especially likely to occur in the eyes in a state of glaucomatous degeneration, but it will at times occur in eyes where from their general appearance and good field of vision we have no reason to expect any degeneration of the intra-ocular vessels. Where it occurs it usually comes on during the operation, pushing forward the vitreous and lens and soon manifesting itself at the incision. It may come in the most skilfully conducted operation without any violence whatever to the eye, but at times it appears to be brought on soon after the operation by the increase of arterial pressure produced by the patient's sitting up in bed, walking back to the ward, or mounting a flight of stairs. How impossible it is with our present methods of diagnosis always to judge as to the likelihood of its occurrence is well shown by a case of Fieuzal[2] where he had severe hemorrhage from one eye, while the fellow-eye had been successfully operated on by him fourteen days previously. That it is sometimes impossible to avoid is well illustrated by a case of Warlomont[3] where he lost one eye by hemorrhage, which he attributed to allowing his patient to walk up stairs after operation. The other eye was operated afterwards by Lebrun with all possible precautions, but was, nevertheless, lost in the same manner. Vomiting after an operation conducted under anæsthetics may also bring it on. It may, however, come on several hours after operation without apparent cause, and Van Duyse[4] reports a case of its occurrence on the second day while the patient was sitting up in bed, while according to Rampoldi[5] it may appear as late as the seventh day. Berry[6] reports a case accompanying vomiting and epileptiform convulsions twelve hours after the operation. Such hemorrhages of the interior of the eye always destroy eyesight and often lead to chronic inflammation and shrinking of the eyeball, even where they have only

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[1] Pechdo, Recueil d'Ophtalmologie, September, 1896, p. 533.
[2] Fieuzal, Bulletin de Soc. Fran. d'Ophth., 1884, p. 141.
[3] Warlomont, Annales d'Oculistique, t. xc. p. 5.
[4] Van Duyse, ibidem, t. cv. p. 112.
[5] Rampoldi, Nagel, Jahresbericht, 1887, S. 400.
[6] Berry, Ophthalmic Review, 1891, p. 95.

emptied the ball of a part of its contents. They rarely cause suppuration of the eyeball (panophthalmitis). Smaller hemorrhages coming on during the healing process are most unfavorable, but not necessarily destructive of eyesight. Arnold[1] reports an interesting case from Haab's clinic where the hemorrhage into the vitreous occurred on the third day and where the little blood-masses were visible with the ophthalmoscope for months afterwards. V = 1/10. Heyl reports from Strawbridge's clinic a case of discission in a ten-year-old boy where, after each of the four discissions, there was hemorrhage into the vitreous, the iris being entirely uninjured. The same may be said of those hemorrhages which occur in the anterior chamber either with or without accompanying inflammation of the iris. Hemorrhages in the anterior chamber during convalescence are more common after the combined operation. Primary hemorrhages which occur after cutting the iris during the operation and which are often sufficient to fill the anterior chamber are not here referred to, only the secondary ones coming on some time after the completion of the operation being here considered. How troublesome and persistent the primary hemorrhages may sometimes be is well shown by a case of Mackenzie[2] where the iris was pricked with the needle used as a cystitome during a flap operation without iridectomy. A slight hemorrhage occurred at the time of operation, but blood escaped subsequently in more considerable quantities, sufficient to stain the bandage and to dry on the face and nose. This oozing continued nearly a week. The eye finally healed, but vision remained very imperfect. Rydel reports an interesting case where on the fifth day after combined cataract extraction blood commenced to flow through the coloboma into the anterior chamber from the region of Schlemm's canal; the hyphæma, which measured three millimetres in height, was finally entirely absorbed, leaving a vision of 20/L. One of the most interesting cases of secondary hemorrhage into the anterior chamber after an operation for combined cataract extraction which the author has ever seen was in the case of a woman fifty years of age, where the operation and healing of the wound left nothing to be desired. On the fourth day, however, while the patient's eye was being gently washed with lukewarm boric acid solution, she started back and closed the lids spasmodically. The wound was not sprung outwardly, but in a few seconds the anterior chamber was absolutely filled with blood. This eventually entirely absorbed, leaving the patient a vision of 20/XXX. Ten years afterwards I again had an opportunity of seeing the patient. The vision was 20/L in the operated eye, but in its fellow there was an over-ripe cataract, and in it she had had an attack of glaucoma a few weeks previously. When hemorrhage occurs at the time of operation or within a few hours after, our remedial measures are, unfortunately, not likely to combat it successfully. Ice compresses have little effect. The raising of

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[1] Arnold, Archiv für Augenheilkunde, 1893, S. 474.

[2] Mackenzie, Diseases of the Eyes, p. 798, London, 1854.

the patient to the erect posture is supposed by some to have a beneficial influence (Jackson, de Schweinitz), while others advise the prompt use of subcutaneous injections of morphia. This has been especially advocated by Dufour. Trousseau reports a case where hemorrhage ceased after the application of a corneo-scleral suture. I have recently seen a case in the practice of my colleague, Dr. Harlan, where the hemorrhage produced a partial detachment of the retina, and having distended the wound protruded between its lips. It ceased spontaneously, the eye still retaining perception of light.

*Frequency, Symptoms, and Pathology of Retrochorioidal Hemorrhage.*—Retrochorioidal hemorrhages are, fortunately, rare. Sattler[1] reports that out of three thousand one hundred and nineteen cataract operations it occurred in 0.119 per cent., and quotes Schiess-Gemuseus as reporting 0.261 per cent. out of one thousand five hundred and thirty cataract operations. Spalding[2] has collected one hundred cases out of literature. When retrochorioidal hemorrhage occurs during or after operation we have usually severe pain in the eye, with hemicrania, nausea, and occasional vomiting, in addition to the symptoms already mentioned,—viz., separation of the lips of the wound, with oozing of blood and protrusion of the iris, lens, and vitreous. White Cooper long ago showed that in most cases of hemorrhage after cataract operations the blood came from the outer and middle layers of the chorioid, and this has been corroborated by many observers since. Lesions of the vascular walls have been found but rarely. Terson reports degeneration of the walls, as does also more recently Bloom,[3] who details the anatomical examination of four such eyes, in all of which phlebitis and periphlebitis were observed. In one case the patient died immediately after the intra-ocular hemorrhage from pulmonary embolism. Wadsworth[4] reports five cases, in one of which the patient died suddenly from apoplexy within a year after the operation.

**Glaucoma after Cataract Operations.**—Glaucoma after cataract operations appears to be of two varieties, one, coming on within a few days after the operation, and more frequently after simple flap operations, arises from the swelling and pressure of retained cortical masses which have accumulated behind the iris; the other, coming on later, is usually the result of the disturbed anatomical relations in the vitreous, ciliary processes, and root of the iris consequent on a capsulotomy, aided by cicatrizing processes which have impaled either the iris or the capsule or both within the wound. Fig. 98 gives the appearance of a wound with cystoid cicatrix and obliteration of the canals of Fontana in a case of glaucoma supervening on the needling of a primarily successful operation for cataract. Pagenstecher relates a case of immediate glaucoma after an extraction in the capsule,

[1] Sattler, Archiv für Ophthalmologie, xlvi., 1, S. 235.
[2] Spalding, Archives of Ophthalmology, xxv., 1, p 92.
[3] Selina Bloom, Archiv für Ophthalmologie, xlvi., 1, S. 184.
[4] Wadsworth, Transactions Amer. Ophth. Soc., 1897, p. 136.

Fig. 98.

Cystoid cicatrix in glaucoma after cataract operation.

where, of course, it could not be due to retained lens-remnants. The glaucoma which often develops after the discission of secondary cataracts may be due either to swelling of the lens-remnants which have been protected from the action of the aqueous by the healed capsule or to inflammatory exudations in the Fontana's spaces. As long ago as 1865 Bowman[1] tells us that glaucomatous tension sometimes comes on after breaking up the vitreous by a needle, and that it is especially likely to occur after needle operations following flap extractions, and less frequent where the incision is accompanied by an iridectomy. In 1869 v. Graefe[2] informs us that glaucoma after cataract operations is by no means rare, more common after discission and reclination, and when occurring after flap extraction due either to swelling of the cortical remnants or to prolapse of the iris. He describes a case where an attack occurred on the day following the operation. Knapp[3] has given us some instructive examples of glaucoma after flap operations which yielded promptly to iridectomy. Treacher Collins[4] reports the examination of twenty-one eyes where glaucoma followed cataract operations. In some instances he found inflammatory exudation due to serous cyclitis to be the cause of the stopping of Fontana's spaces, while in most instances the closure of the filtration angle was due to impalement of either the iris, the capsule, or the vitreous in the wound. In four of these cases in which iridectomy had been effectual in arresting the glaucoma, he found that in two the iris had been torn from the ciliary body at their junction, thus opening the filtration angle at this point; in one, although a stump of the iris was left, the angle was open in the coloboma and elsewhere; while in the fourth there was a fistula of the sclerotic in the wound through which the aqueous escaped into the subconjunctival space.

*Treatment.*—In all cases instillations of eserine or pilocarpine are advisable. Pagenstecher[5] recommends, in addition, the use of hot dry compresses and the internal administration of sodium salicylate in doses of from thirty to seventy-five grains daily. If these measures fail, resort must be had to iridectomy or sclerotomy. When the anterior chamber is opened and the aqueous escapes, the loop of vitreous in the pupillary space will at times come forward and become entangled in the wound, and when this occurs it makes the correct excision of the iris difficult.

**Detachment of the Retina.**—Detachment of the retina when supervening after cataract operations is usually the result of manifest cyclitic processes. An interesting case occurred in the author's practice where sudden detachment of the retina came on in an eye previously operated on for cataract while the patient was using a pickaxe to dig a ditch. The eye had been quiet and useful for five years previous. In such cases it may be

[1] Bowman, R. L. O. H. Rep., 1865, p. 365.
[2] V. Graefe, Archiv für Ophthalmologie, xv., 3, S. 221.
[3] Knapp, Archives of Ophthalmology, xxi. p. 558.
[4] Treacher Collins, Lancet, December, 1894.
[5] Pagenstecher, Klinische Monatsblätter für Augenheilkunde, 1895, S. 139.

doubted whether the previous operation for cataract had any direct influence in producing the result. Hirschberg relates an interesting case where, in a patient with complete detachment on one side and partial detachment with hemorrhages on the other, cataract formed in the eye which retained some visual power. The cataract was removed with apparently good results as regards the detachment, since twelve years later the patient could get about and employ herself at coarse work. The field was 40° above, 50° down, 35° in, and 40° out.

**Colored Vision after Cataract Operations.**—*Kyanopsia.*—Patients will sometimes state immediately after the extraction of a cataract that there is a blue haze in front of them and that objects appear blue. Becker has pointed out that this is probably due to the diffusion of light caused by thin layers of cortical which still remain in the pupillary space. The blue vision disappears with the absorption of the cortical masses. This appearance has been interpreted by others as a phenomenon due to retinal fatigue, believing that the eye, having long been saturated by the yellow light coming through the nucleus of the lens, sees the complementary color after its removal. Burnett has recently maintained this view, and proposed for it the name kyanopsia.

*Erythropsia.*—It is of occasional occurrence that persons who have had no operation or inflammation of the eyes see all objects with a red tinge of varying intensity. These cases appear to belong to two classes, one due to prolonged exposure of the eyes to an intense light, the other probably due to some irritation of the cortical sight centres. Its occurrence is so frequent after cataract operations that it looks as if it were then due to local causes and standing in direct causal connection with the operation. Van Millingen has given us an interesting account of two attacks in his own person, and in each case it was probably central in its origin and due to fever. In the first instance it came on during an attack of tonsillitis, with a temperature of 38°–39.5° C. All candle flames appeared of a bright scarlet red, and this phenomenon lasted seven days. In the second, during an attack of scarlet fever, with a temperature of 40.3°, white objects by daylight appeared yellowish, while at night candle flames were of a bright carmine red. This phenomenon disappeared with the subsidence of the temperature. Cleeman[1] has recorded an interesting case where a bright-red spot appeared in the centre of the field of vision, which was seen most distinctly in looking at lights and white surfaces. It came in an apparently healthy individual, where the eye-ground on the following day was found to be normal, and where there was no possibility of any retrobulbar neuritis, due either to tobacco, alcohol, or any other toxic agent. The vision remained normal in this eye for years subsequent to this transient central red vision. Other instances of red vision have been observed in migraine, in acute mania, in religious mania, in zoopsia, and as preceding the epilep-

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[1] University Medical Magizine, vol. vii. p. 403.

tic aura. On the other hand, it would at times seem that change in the retina or optic nerve might be the cause. Thus, Berger[1] records a case which followed a blow on the cheek; Kubli,[2] one where there was hemeralopia and "undoubted retinal affection;" Szili[3] relates an instance where the red vision was accompanied by double papillitis, and lasted until there was atrophy of the nerves. In some instances it has been attributed to the glare of light entering the eye after an iridectomy, and cases are reported after iridectomy for glaucoma, and many after the combined operation for cataract. Cases are also reported after simple dilatation of the pupil by a mydriatic (Valude[4]). Mayerhausen,[5] in a case of posterior cortical cataract in a microphthalmic eye, produced red vision by instillation of atropine, and some time after a second instillation produced a similar result. Hirschler[6] had a case where the erythropsia came on during a visit to the country and disappeared on returning to the city, while Dimmer's[7] case had red vision in the house, but which disappeared when the patient was out-of-doors, and Katzarow[8] had a case where the red vision was most noticeable by moonlight. Steinheim[9] records an instance in an hysterical patient with a coloboma, where the red vision was most marked at twilight. In Dimmer's case there was no great dilatation of the pupil, the patient having been operated on by discission, as was also the case in the instance reported by Westhoff.[10] In the latter case the patient sorted Holmgren's wools properly, but persisted in calling white rose-colored. In many cases coming on after operation the accompanying symptoms make it probable that they are nevertheless due to central causes. Thus, Hilbert[11] observed erythropsia coming on a year after extraction. It lasted one hour, and was accompanied by intense mental excitement; two days later it recurred under similar circumstances. The author has never seen but one well-marked case. It occurred in a man of sixty-five years in the third week after the combined operation for cataract. The eye was quiet, the vision good, and no pathological appearances could be detected in the retina or chorioid with the ophthalmoscope. The symptom disappeared in the course of a few weeks, and this, I believe, is the usual course of such cases. Galezowski,[12] however, reports five cases where the red vision persisted to an annoying extent for a long time. Fuchs, who has studied the subject carefully, says that

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[1] Berger, Centralblatt für prakt. Augenheilkunde, 1885, S. 140.
[2] Kubli, Nagel, Jahresbericht, 1887, S. 407.
[3] Szili, Klinische Monatsblätter für Augenheilkunde, 1886, S. 259.
[4] Valude, Archives d'Ophtalmologie, 1888, viii. p. 130.
[5] Mayerhausen, Wiener med. Presse, 1882, No. 42.
[6] Hirschler, Wiener med. Wochenschrift, 1883, No. 4.
[7] Dimmer, ibidem, 1883, No. 15.
[8] Katzarow, Nagel, Jahresbericht, 1884, S 580.
[9] Steinheim, ibidem, 1884, S. 580.
[10] Westhoff, ibidem, 1888, S. 351.
[11] Hilbert, Klinische Monatsblätter für Augenheilkunde, 1886, S. 483.
[12] Galezowski, Recueil d'Ophtalmologie, 1879, p. 584.

he has almost invariably been able to produce erythropsia in his cataract patients by sending them to walk in the sunlit snow on a clear winter's day. He thinks that aphakic eyes are more disposed to it, because the lens has fluorescent properties, and, moreover, in old people is yellow. It therefore, while present, prevents the entrance into the eye of great numbers of the rays of short vibrations at the violet end of the spectrum, and its absence, allowing the free entrance of these, gives a much better chance of retinal exhaustion. We are also indebted to Fuchs[1] for a minute study of the phenomena and causes of purple vision in normal eyes. In his own eyes, Fuchs readily brings on erythropsia by long walks over snow-fields on high mountains, and upon entering some dark shelter hut immediately thereafter sees all black objects green and white ones purple, the purple being complementary to the spectrum green, which lies on both sides of the line F. Both green and purple are less brilliant at the fovea than five degrees from it. In Fuchs the erythropsia extended from fifty to sixty degrees to the temporal side and from twenty-five to thirty-five degrees to the nasal side, while in Koster it extended nearly to the limits of the temporal visual field. Fuchs maintains that these appearances cannot be the positive after-images of white light, because after a walk on the snow, the eyes having been protected by colored glass, the usual colored after-images manifest themselves, and when these have again disappeared, the typical erythropsia comes on as usual. He supposes that the cones are then able to perceive the retinal red, as it is regenerated after exhaustion by bright light in the surrounding retinal rods. Schulek[2] has also experimented a good deal in producing erythropsia. He has produced it in his own (normal) eyes by looking for a time at the ultra-violet end of the spectrum, and even when he had thus wearied only a small part of his retina, nevertheless the entire retina became erythropic. He adduces a number of cataract-operated people in whom it occurred, and gives an especially interesting case in the person of Dr. Hirschler, in whom it came on for the first time five months after the operation, following exposure to bright light out-of-doors, where it as usual showed itself most distinctly in passing from a strong to a feeble light. Snellen[3] believes that erythropsia is an after-image and contrast-effect of the red-purple light, which in case of exposure to intense glare is transmitted through the translucent and vascular eyelids to the retina. Hirschler, who suffered from it in his own aphakic eyes, found it to be associated with hemeralopia, and Fuchs found, by testing erythropic aphakic eyes with Foerster's light-perception measure, that the operated eye always took longer to accommodate itself to the comparative darkness and to be able to count the stripes in the photometer than did the unoperated fellow-eye. The great rarity of this phenomenon in the

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[1] Fuchs, Erythropsie, Archiv für Ophthalmologie, xlii., 4, S. 207–293.
[2] Schulek, Ungarische Beiträge zur Augenheilkunde, Bd. i. S. 108.
[3] Snellen, Archiv für Ophthalmologie, 1897, xliv., 1, S. 19.

author's practice is probably due to the fact that he has always insisted on the use of London smoked glasses for many weeks after operation whenever the patient went out of-doors or was facing a bright light.

**On the Delayed Union of Wounds made in Cataract Operations.**—The normal course of healing of wounds made in cataract operations has already been described, but in some instances this fails to occur. Delayed union should lead us to look carefully for some local cause, such as the impalement of iris tissue, of vitreous, or of capsule in the wound, but where careful search with magnifying-glass and oblique light fails to reveal any local hinderance, we may still have long delay in the closure of the wound and the re-establishment of the anterior chamber. Where the anterior chamber does not reform in spite of apparent closure of the wound, iridectomy has been recommended, and appears in some instances to bring about prompt and firm healing of the wound. Mackenzie[1] reports a case where the wound did not unite for nine weeks, and another which opened on the fourteenth day, closing again on the day following. He also quotes Raleigh as giving three cases in which, although there was no escape of vitreous, delayed union occurred. In one of them the cornea became flaccid, dull, and opaque, and the eye shrank ; the two other cases recovered under the use of pepper poultices to the lids and of nitrate of silver to the conjunctival sac. Jacobson gives an instance of delayed union of over four weeks' duration. There was blepharospasm, and aqueous escaped as often as the bandage was left off. Jacobson then kept the eye closed by a compress bandage for ten consecutive days and nights, after which time the wound remained closed. Despagnet[2] reports a case occurring in a woman eighty years of age, where for eight weeks no closure of the wound occurred. At this date hypopyon set in, which was evacuated by puncture of the anterior chamber, a piece of the iris being at the same time excised. The wound commenced to heal on the ninth day after the iridectomy, and the eye soon recovered. Trousseau,[3] in a patient aged sixty-four, with emphysema of the lungs and Bright's disease, failed to obtain union of the flap. On the fifteenth day the patient insisted on removing the bandage, and on the thirtieth day the lips of the wound were united, except at the middle of the cut, where a little aqueous oozed out each time the patient coughed. This fistula remained permanent, and two months later the patient, having refused to submit to an operation of iridectomy, was discharged. Vignes has reported two cases, in one of which the wound remained open ten days and in the other twenty days. Barck has published three cases of delayed union, two in which the healing took place after two weeks with good results, while suppuration of the cornea set in on the eighteenth day in the third. Koenig has seen delayed union for

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[1] Mackenzie, Diseases of the Eye, London, 1854, p. 804.

[2] Despagnet, Archives d'Ophtalmologie, Avril, 1897, p. 267.

[3] Trousseau, La France Médicale, Avril, 1894.

twenty days. Bourgeois and Jocqs recite two cases, one lasting two, the other three weeks, both of which they attribute to entropion.

Harlan[1] has lately written an instructive paper on this subject, and adds two cases to those already published. One of these, after non-union of two weeks' duration, healed under a continuation of the compress bandage accompanied by improvement in the general health. In the other there was a preliminary iridectomy and non-union for three weeks, when the application of the mitigated silver stick to the wound caused prompt union with excellent and lasting vision. The same author also gives the date of closure of the wound in five hundred consecutive cases of cataract operated by various surgeons at the Wills Hospital. In four hundred and seventy-four of these the anterior chamber was re-established in less than five days, and in the great majority of instances within twenty-four hours. In the remaining twenty-six cases there was delayed union, and in nine cases the wound remained open for five days, in two cases it remained open for seven days, in three cases for eleven days, in two cases for twelve days, in one case for thirteen days, in one case for fifteen days, in one case for sixteen days, in one case for seventeen days, in one case for nineteen days, and in one case for twenty days. Of these twenty-six cases of delayed union fifteen were in simple and eleven in combined extractions. In twenty there was no complication during the operation, but of the remaining six there were four with prolapse of the iris and two with prolapse of the vitreous into the wound. These statistics were obtained by the resident surgeon, Dr. C. J. Kistler, under Dr. Harlan's supervision, and are doubtless correct, although the cases of delayed union in this series have been vastly more frequent than those in the author's own practice. The writer has seen three weeks elapse without any union of the wound without untoward occurrence and with ultimate good result, and another in which after two weeks rapid union occurred (as in the case above related by Harlan) after the use of a fine point of mitigated stick of silver nitrate drawn gently and rapidly over the wound.

*Causes of Delayed Union.*—As regards the constitutional condition of the patient, it undoubtedly occurs at times in patients who are neither albuminuric nor diabetic, and in whom no marked dyscrasia is demonstrable. As was mentioned at the commencement of this heading, we are considering only cases where there is neither lens-matter, vitreous, nor capsule in the wound, and excluding, of course, also all in which increased or glaucomatous tension is present. In the latter cases, as was justly remarked by Becker, we are lucky if we get off with a cystoid cicatrix. Excluding then these cases, and considering those only where there is good coaptation of the wound, but no firm adhesion between its lips, it would seem that an important *rôle* is played by the faulty nutrition of the tissue, and that we have non-union here as we do occasionally in simple fracture of the bones. Where

[1] Harlan, Delayed Union after Cataract Operation, Trans. Amer. Ophth. Soc., 1898.

there is feeble union this may be destroyed by spasmodic action of the orbicularis, and of course by its action, where no reparative action has taken place, the wound may be still more readily made to gape. This is well illustrated by the case of Jacobson (above quoted), where recurrent blepharospasm kept the wound open, and also by those reported by Bourgeois and Jocqs, where there was entropion and where healing promptly took place when the entropion was cured, in one instance by the removal of the bandage, in the other by operation.

*Treatment.*—Iridectomy has been proposed as a cure in all cases of non-union. As striking examples of its good effect may be cited the cases of Valude and of Terson. In the former the wound had remained open for several weeks, and it closed within twenty-four hours after the performance of the iridectomy. In the second, the anterior chamber had remained open for a month and a half, closing in two days after the performance of this operation. Harlan and Norris each report prompt healing after the careful use of the mitigated stick of silver nitrate on the lips of the wound. Vacher advocates the use of tincture of iodine. All these surgical procedures should be reserved for obstinate cases, where after two or three weeks we have failed to obtain union. The majority of cases will heal by rest, with daily bandaging and careful cleansing of the conjunctival sac, aided by improvement of the nutrition and of the general health.

# AMETROPIA: ITS ETIOLOGY, COURSE, AND TREATMENT.

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## PART I.

## GENERAL CONSIDERATIONS.

### INTRODUCTION.

*Difference between the Eye of the Savage and that of Civilized Man.*—When it is considered that the eye of the human savage can bring exceedingly small pencils of light to a focus on its sentient sheet, and thus afford its possessor the power to recognize objects at greater distances than civilized man, it must be conceded that this difference is in part dependent on peculiarities of living to which the two types of mankind are subjected.

What this difference consists in becomes manifest when the modes of livelihood of these two classes are contrasted. The savage is a man of nature, who gains his sustenance in almost the same fashion as does the prey he seeks. He gazes for brief periods of time at near things that require but little effort to see, and he constantly educates his powers of visual penetration for distant objects both by day and by night. For him such a type of vision is a necessity; without an eye that can subserve such a purpose he is useless.

On the contrary, civilized man is gathered into communities from which the greater number of out-of-door pursuits are for the most part precluded. He obtains his living in such environments by the constant use of his delicately contrived organs, such as the eyes. His visual organ must be largely adapted for near work: without such an apparatus he is debarred from many of his duties.

When these two series of facts have been considered, it will be admitted that the wear and the tear of the visual organs in the latter type of mankind are the greater. In other words, there are two types of man, each employing almost the same grade of visual apparatus to such different degrees that in the first there is little or no strain brought upon it, while in the second the organ is so often abused that there is structural impairment.

These results in the latter type of man are the physical penalties for the possession of increased mental force: results that are the consequences which civilized man must accept in order that he may enjoy the fruits of civilization and culture.

The conditions manifest themselves in many places. They not only are found in professional walks of life, but also are evident in the preparation that is necessary to render such walks remunerative. They are seen in the artisan, whose visual apparatus is tried by the delicate manipulations and the accuracies of vision that are necessary for the proper performance of his work. The seamstress, the weaver, and even the housewife, in their several vocations, feel the strain of over-exertion of visual function, and the taxation of the ocular tissue employed. In addition, the new-born infant may have given to it an inheritance of unstable or inflamed material that cannot withstand any ordinary amount of use, thus rendering it less valuable as an individual and more prone to transmit a harmful taint.

What does this signify? It means that there is either a malformation or an inflammation of congenital type, or that there is an acquired fault; each expressing itself by physical or physiological impairment.

*Flattened Eyeball of Man.*—Examination of great numbers of similar cases to form correct averages has shown that the ordinary shape of the unemployed eye of the human species is that of a vertically placed spheroid which is so situated as to present its short diameter to the entering rays of light. As many types of mankind evince differences in cephalic configuration and facial contour, so similar peculiarities of structure exist in the visual organs.

*Normal Eye and Emmetropic Eye.*—If the flattened eyeball be the average visual organ in man, the question arises, what must be assumed as the normal one in contradistinction to what is known as the standard or "emmetropic" eye? By "normal eye" is not meant the findings of the mathematical formulæ of the arithmetician, which give ratios of supposed dioptric perfectness that are used to specify the emmetropic organ. The "normal eye" is not designated by the dividing line between the convex and the concave correcting lenses that has been assigned as the expression of an emmetropic ideal by the working clinician. Neither is the "normal eye" the result of the solution of the geometric and trignometric problems that offer some optical theorists the assumption of the condition of absolute sphericity.

Just as with any other eye, the existence of such a normal eye is dependent on the freedom of the structures from disease, in association with an undisturbing physiologic action giving as near a normal visual result for both near and far as possible. Provided that these conditions be present, it is of no consequence what the shape or what the size of the organ may be. If it be healthy and if it be acting properly, it is normal.

Totally different from this is the emmetropic standard—the perfect, the ideal, the ever-to-be-sought-for organ: the one by which so-termed perfec-

tion of visual result is obtained. Unfortunately, in but a low percentage of cases has such an ideal of the mathematician, the optical theorist, and the physiologic ophthalmologist any existence in the human form. In fact, if the emmetropic eye be considered, as it scientifically should be, as a perfect visual mechanism, in which parallel rays of light fall in exact incidence on the part of its sentient sheet that is devoted to direct vision when the organ is at rest, it is doubtful that it has any existence at all. The emmetropic eye, as it is clinically seen at times, is but a temporary condition in what is known as lessening ametropia: the transitional stage between hypermetropia and myopia. It is an organ that for a brief season focuses distant parallel rays on its foveal plane without muscular effort, giving distinct images of distant objects. It is a refractive halt, as it were, in an asthenopic eye, a complaining organ, an irritated or inflamed one that needs treatment.

In reality, emmetropia should be understood as only a standard form with which all ametropia may be compared, all departures from emmetropia being known under the generic term of ametropia. The perfection of form in the working human eye, and then admittedly only in the antero-posterior diameter along and about the visual axis, is as rare as a symmetric face or a perfect human figure. In the ocular bulb, any physiologic act is so disturbed by what would be a minor discrepancy in another organ or association of features or parts of the body, that pronounced optical error with grave consequences to delicate structures is too frequently the result.

Practically, the ideal or the emmetropic standard should always be sought for, even though it is a theoretic stand-point that cannot be gained. The condition artificially obtained, however, is not always the best for the well being of the organ. For example, the ideal dioptric equivalent reached in a single visual organ may be at variance with its fellow: again, a perfect vision obtained by neutralizing lens s may be fraught with increased physiologic action that is detrimental to the disturbed physical material.

*Cyclopia.*—Should the human visual apparatus have terminated in a single eyeball, the problem would be easy of solution. Instead, there are two end-organs, each of which receives a sensory impression of such a character as to permit the compound impression to be combined into a single perception. This, which is known as binocular single vision, means the simultaneous reception of two dissimilar macular impressions on the two cuneal regions in the occipital cortex, from which positions a single mental picture of the associated images is formed. No matter in what part of the combined visual fields the external objects may be situated, the rule, as modified by circumstances, holds good.

## AMETROPIA AND ASTHENOPIA.

Though ametropia is as wide-spread as the world itself, yet asthenopia is the bane of the civilized minority. In this class of subjects the subconscious acts that have been learned so early in life give way by reason of

some disturbance having its beginning either in the organ itself or in the general system. Such disturbing factors render the necessary automatism so irregular as not only to provoke improper physiologic result, but also to help fasten injurious consequences on the structures that are immediately connected with the work itself, as well as on those belonging to more or less related organs.

As illustrative, suppose that a ciliary muscle is brought into constant use by an inadequacy of the focussing power of the refractive apparatus of the eye. From such a persistent action, not only do local pathologic changes take place in the visual apparatus, but general disturbances may appear. Suppose such an overaction, or possibly an unequal overaction of the two ciliary muscles, should bring a want of equilibration of the two series of extra-ocular muscle-groupings into play, an additional causative factor for eye-strain is produced. Moreover, suppose that, through faulty corneal curvature, irregular lenticular play, or disturbed muscle-balance, etc., an improper mental picture is produced, how much more labor is given to the visual act, and how increasedly pronounced become the asthenopic factors!

Asthenopia or difficult vision is dependent on many interrelated and interdependent conditions. Should, for example, the combined coefficients of the refractive media be imperfect, compensatory physiologic processes, producing a train of disturbing symptoms best known under the term *ametropic asthenopia*, may make their appearance; or, if the two eyeballs be so dissimilar as to give rise to imperfect muscle-equilibrium, with irregular muscular movements, an additional series of distressing and ofttimes injurious symptoms, that can be designated under the term *heterophoric asthenopia*, may be provoked.

Here the symptomatic terms *muscular asthenopia* and *accommodative asthenopia* have been discarded. This has been done because the first simply designates that there is a weak sight which is caused by faulty muscle-action, and the second denotes that there is a weak sight which is caused by an improper physiologic action; the two being at variance with their real meanings, and the second included in the first.

*Ametropic Asthenopia.*—This form of eye-strain includes in its etiology both the sensory and the motor disturbances that have their origin in badly shaped eyeballs and faultily constructed or improperly acting dioptric media.

*Heterophoric Asthenopia.*—This variety of asthenopia has for its causative factors idiopathic or functional errors in muscle-equilibrium that may be independent of the shape of the eyeball and the condition of the refractive elements.

Heterophoric asthenopia is not so rare as may be supposed. Be the ametropia never so slight or undisturbing, or even if emmetropia be found, asthenopic symptoms of heterophoric type may be present. Moreover, idiopathic or primary heterophoria, particularly hyperphoria, may not only

produce a heterophoric asthenopia, but also provoke an ametropic one from a latent ametropia which would not have been brought into evidence had there been a normal equilibrium and action of the ocular muscles.

Considered broadly, asthenopia or eye-strain is a complex symptom-grouping. As a rule, it is expressive of the combined workings of the two ocular end-bulbs and their accessories. Granted a manifest esophoria of one or possibly two degrees as the normal average for combined distant vision, each individual, and even the same individual under different circumstances and during varying states of fatigue and health, has dissimilar degrees of physiologic tone and power given to his ocular muscle-series during both far and near vision. What may be the normal state of any visual apparatus of to-day need not be that of the morrow, while the amount of ametropia that in some instances must be considered abnormal may ofttimes bear a sufficiently harmonious relationship to the muscular portion of the visual apparatus to produce temporarily comfortable binocular vision. Orthophoria, like emmetropia, is a theoretic, a mathematic, and a geometric standard, and is no more expressive of normality of tissue and harmony of action than the latter. Normality can be assumed, no matter what inequality may exist, as long as the two series of ocular muscle-groupings are in balance, and continue so during activity.

The problem would be different and less complicated if the human species were cyclopic, but there are two eyes that are brought into intimate association in a subject of unstable physical equilibrium, and hence of changeable physiologic action.

*Asthenopia and Reflex Neuroses.*—It is the heterophoric type of asthenopia, particularly the hyperphoric varieties of minor degree, more than it is the ametropic one, which gives rise to so many reflex neuroses. It is in this type of disorder that dizziness, gastric disturbances—with urticaria, for example—vertigo, migraine, nervous irritability, mental confusion, insomnia, etc., all of which are so prone to interfere with general nutrition, come into existence. In this category are to be found the many changes of vaso-motor type. In this grouping are to be seen the characteristics of the vicious circle of cause and effect: eye-strain, with its pain and nervous disturbances, producing interference with assimilation and nutrition, which in its turn so reduces the general physical condition as to induce an increase in the asthenopia.

While this is true in regard to nerve-storms that are produced by heterophoria (as also ametropia), it must be remembered that disturbed motor impulses which cause reflex disorder about the two eyes may compel so rapid a discontinuance of eye-work that the more distant reflexes may never be given opportunity to manifest themselves. Furthermore, the general nerve-tone of the subject can be so strong that any weak excitation by a low-grade causative factor may, unless constantly repeated, like the continual dropping of water on a stone, never make an impression.

Just as it is true that ametropic and heterophoric asthenopias give their

possessors far-reaching and injurious symptoms that can be removed in no other way than by correction of the local defects, so it is certain that general disturbances, even when centred about the most remote and seemingly unconnected organs, may provoke ocular symptoms that are irremediable except by a therapy that is directed towards the casual factors. These facts render it certain that asthenopia, which need not be present with the ametropia and heterophoria of the sthenic, is sooner brought into existence in the neurotic, the toxæmic, and the anæmic subject; and it is in this last class that the minor degrees of ametropia and heterophoria so early manifest their presence.

In fact, in some such cases many of the functional nervous diseases and some of the more permanent morbid processes of the cerebral cortex itself, known under the names of chorea, epilepsy, etc., may in a measure be indirectly dependent on disturbed binocular action. Closer clinical research and broader unbiased generalization, more improved methods of precision, less few and more carefully made autopsies, better prepared microscopic material, and finer differentiation of structural change, must, however, be made before any certainty of cause and effect can be offered by the conscientious and scientific physician.

*Neurasthenia as a Cause of Eye-Strain.*—Neurasthenia plays a more important rôle in the production of asthenopia than is usually taught. Functional pareses of the motor apparatus of the two associated ocular bulbs, that are ever changing and even infinitesimal at times, frequently evidence themselves by varying degrees of pupillary dilatation and irregularity of iris-motility during near-focussing. Sensory fatigue, as expressed by distressing contrast-pictures and confusing after-images, is often present. Vaso-motor disturbances, both subjective and objective, such as increased lacrymation, undue congestion, sensation of foreign material in the conjunctival culs-de-sac from want of tone and localized dilatation of the vascular walls, with a sense of weight and a tendency to drooping of the upper eyelids, may be present; while, to make the ocular symptoms more complex, painful and even harmful hyperæsthesias and irregular spasmodic actions frequently manifest themselves.

It is the minor and the unequal degrees of heterophoria (particularly hyperphoria) that produce the most disturbing reflex symptoms. This is especially so if there be some peculiarity of angle of muscle-deviation or faulty muscular tendency. Here the inter-association of the eyes, the character of the tasks given to them, the health, and the gross anatomic conformation of the subject, all exert their individual and their combined influences.

*Pain as an Expression of Eye-Strain.*—Neuralgia, the functional expression of a disturbed nerve, is one of the most common signs of ametropia. Situated in varying positions in the course of the trifacial and the second cervical groupings, it not only manifests itself as the direct result of the use of the eyes, but appears more readily when the system is below par,—

during convalescence, menstruation, and temporary exhaustion, and particularly when some blood-dyscrasia, such as gout or rheumatism, is present. The character of the ametropia, its inequality, and its degree, all become potent factors in the intensity and the frequency of the occurrence of cephalalgia.

As empirically determined, without resort to statistics, which at the best are so uncertain and idiocratic, it may be stated that low grades of compound hypermetropic astigmatism, mixed astigmatism (with that in which the hypermetropia is preponderant coming first), followed by compound myopic astigmatism, hypermetropia, simple hypermetropic astigmatism, simple myopic astigmatism, and myopia, are, in the order here given, the ametropic types producing the greatest amounts of headache. Anisometropia and antimetropia, particularly the former, are very apt to increase the frequency of cephalalgia.

As yet, the differential diagnosis between the cephalalgia of ametropia and that of heterophoria is uncertain, though it is probable that when the pains are associated with dizziness and "carsickness," for example, abnormal muscle-balance is the more to blame. As a rule, however, the two conditions are associated and interdependent.

*Local Reflex Eye-Strain.*—At times a type of asthenopia may be seen which has been produced by conjunctival and palpebral irritation and inflammation. Rarely uncomplicated, more often interrelated, and frequently associated with gross disturbances, such as pterygia, chalazia, and abnormal adhesion between the lid and the eyeball, the eye-strain results as a consequence instead of being a cause. Whether naso-pharyngeal disturbances can be included in this category as a direct influence is uncertain, although it is positive that the removal of adenoid vegetations, with the consequent betterment of the mucous surfaces of the lacrymal apparatus and conjunctiva, has indirectly relieved cases in which the correction of the ametropia and the heterophoria has availed little or nothing. Nasal reflexes from Schneiderian-membrane troubles, hypertrophic turbinates, etc., must also be considered in the position of causal relations, particularly if the main asthenopic symptoms and the nasal disturbances be unilateral in type. So, too, with dental caries. In this connection, it may be pertinent to suggest that many subjects suffering from frontal neuralgia and complaining of eye-strain on rising in the morning may have their symptoms in a measure dependent on congestion of the nasal cavities and passages, from lid, conjunctival, and lacrymal irritation that has been provoked the evening before by undue or improper use of the eyes during a period of prolonged near work.

*Spastic and Overacting Types of Heterophoric Asthenopia.*—In the spastic variety or the overacting type of heterophoric asthenopia, particularly when there is hyperphoria and the interior muscle-groupings are also affected, migrainous attacks become quite common. In forced or overacting exophoria, the more general signs, such as vertigo, incoördination, mental peculiarities, and even insomnia and neurasthenia are noticeable.

Undue action of the interior muscles (the sphincter irides and the ciliary) must, being supplied by the third nerve, affect all of the related exterior muscles (the inferior oblique, with the superior, the inferior, and the internal recti muscles), and not, as is so generally taught, a single muscle. This increased functional exertion produces a departure of the globe from orthophoric equilibrium to all those forms of heterophoria and heterotropia that are the results of overaction of the combined series of exterior ocular muscles which are governed by the third nerve. As a natural result of this, minor amounts of deviation (heterophoria) are, as a rule, produced by the relatively low degrees of imbalance, and the major variations (heterotropia) are brought into existence by the relatively high degrees of want of equilibrium. As the inclination of the two globes inward and upward is the most pronounced direction of movement that is generally obtained by this series of muscles, the same relative angle of deviation will occur when these muscles are made equally to overact their unequal powers.

*Paretic or Underacting Forms of Heterophoric Asthenopia.* — In the paretic form or the underacting variety of heterophoric asthenopia, that is found so frequently in the asthenic, and which is in part usually dependent on weakness in the muscles under third-nerve impulse, the subjective symptoms present themselves principally as cephalagias that may vary in all manner of ways from mere frontal or fronto-occipital pain produced by prolonged or undue simultaneous use of the two eyes to the suboccipital and cervico-occipital forms of neuralgia that may appear at any time. To these expressions must be added those peculiarities of mentation that will be referred to later. The reason why this form of heterophoric asthenopia is early present when the vertical deviations are impaired is that the muscles governing such movements are relatively the weakest, and have more limited actions, the greatest disturbance of this type always being the earliest found in the positions of the weakest groupings or combinations of muscles.

Another variety of this form of asthenopia, one of mixed type (both the underacting and the paretic), is that in which both the extrinsic and the intrinsic muscles of the two eyes are weak and inefficient as a part of some general debility. In this form of weak sight there is a subnormal ciliary action, which is associated with slightly dilated pupils, and an exophoria during near vision. This is the most frequent form of disorder, in which, for characteristic symptoms, there are both ametropic and heterophoric disturbances with a tendency to diffusion circles. Here, in contradistinction to the series of symptoms that are produced by overaction of the ocular muscle-groupings, there are not only an inability for prolonged focussing and an imperfect binocular fixation, but also visual confusion, dizziness, and gastric disorder.

When the paretic form of asthenopia is of systemic origin—be it either temporary from neurasthenia or permanent from senility, for example—not only are the muscles that are supplied by the oculo-motor nerve weakened,

but also those that are innervated by the fourth and the sixth nerves. In this form of disorder, the entire motor-groupings become lowered in action, as can be shown by means of myometric tests; and a lessened and disturbing muscle-action will be one of the results.

*Climate as a Cause of Asthenopia.*—Climate is one of the most potent of etiologic factors in the production of asthenopia. A glance at the indolence of those who, living in tropical and subtropical situations, are content to exist for many months of the year almost as the tree beneath which they rest to escape the heat and glare of the noon-day sun, will explain this. Here there is an absence of work—either mental or manual. Here nature in her generosity bids but to stretch forth the hand to pluck the fruit that furnishes both bread and water. Appreciate the unnecessity for apparel except for decency's sake. Realize the uselessness of permanent habitation where, as for the birds and the beasts, houses are everywhere. Here is man untutored and unread. Here he is simply a part of animation, with his few wants and his fewer desires, passing a brief holiday-time, to perish just as do the plant, the bird, and the beast, that to him have been at once his friends, his enemies, and his existence. Here, in such a life, in which there is no tendency to close or applied work; where to live is enough; and in which there are but few hereditary tendencies to ocular change and no disturbing employments: here must be most frequently found the normal and the uninjured flattened eyeball of man.

How different is life for the dwellers of the rigorous climates of the North and the South! Fighting for every morsel of food to eat and working for each drop of water to drink; necessarily covered with protective raiment of many forms during the entire year; constantly trafficking and bartering for materials that cannot be grown or made in their own countries; and compelled to be surrounded with permanent dwellings to shield themselves and their families from cruel beasts, more cruel man, and most cruel nature: is it any wonder, when it is realized that to acquire the amount of knowledge and technique that are necessary to understand the duties which are required to preserve their existence, that their lives must be one ceaseless toil, and that every organ must be gauged to its utmost working capacity! From early childhood, schools must be gone to; during youth, colleges for both theoretical and practical knowledge must be attended; and during manhood, the application of such information must be incessantly applied. Moreover, for those who aspire to be the most successful in any one of the many pursuits in which they are engaged, increased mental and monetary capital above that of their fellows must be amassed; less skilled, perfunctory, and routine labor must be bought; competitors must be overthrown; and every detail of business must be unceasingly inquired into, corrected, bettered, replanned, and rearranged. In other words, in such a struggle for existence, man is undergoing constant physical labor. To live in such an environment, he is compelled to endure all of the evil consequences of wear and tear of structural organs; and

hence, here in such a life, it is that the eye—the most useful and the most delicate end-organ in the organism—is unremittingly and ofttimes injuriously brought into play. So, too, with many other factors; each plays its etiologic part.

*Interrelation of the Eye and the General System.*—As the complicated visual apparatus works harmoniously and rests adequately, it will exercise a healthful influence on many of the distantly placed organs. So, conversely, with the other organs on the ocular apparatus: every jot of normal activity in them and every tittle of repose they obtain will help to bring healthful vigor and adequate nourishment to those portions of the body that are intended for visual purposes. There is a circle of events or circumstances for weal or for woe, as it were; the one being both the cause and the effect of the other.

What influence an optical error, producing imperfect and at times erroneous perceptions and faulty conceptions, has not only upon the possessor's aims, his ambitions, and his very conduct of life, but also on the formation of his character and the moulding of proclivities and tastes which necessarily determine his usefulness as a bread-winner and his importance to the community, is not ordinarily appreciated.

*Correction of Ametropia.*—Not all ametropia, however, needs lenticular or prismatic correction. A moment's thought of the Arabs and the Moors, who are able to ken the smallest form of animation across immense and almost colorless sweeps of arid waste, where their European neighbors cannot discern anything, will show this; as here among these Barbarians the graphic symbols of the stars of the firmament are supplied with the radii of astigmatism. A knowledge of the power of the American Indians, who, without warning, can recognize the minutest change of movement in a distant landscape which is as nothing to their white brothers, will at once evidence this, and yet repeated examinations have shown them to be astigmatics of no mean degree. A study of the contrast between the unaided vision of the unaliened tribes of mountain-dwellers, such as the pure Mexicans, living their lives amid broad vistas and in vast expanses of country, with that of the bespectacled city-dwelling Germans of Northern Europe, who with their books, their magnifying lenses, and their wonders of intricate and delicate mechanism and handicraft, are compelled to forego a thousand beauties of nature and are ofttimes forced to walk alone with their own marvellous creations, will immediately reveal this, and yet the banners and the decorative work of the former display the radial star.

*Conclusion.*—It will thus be seen that, although theoretically correct, yet practically it is not possible to consider the functions of the visual organs as dominated by any fixed mathematical laws. It is merely the unconscious action of related healthy working tissues that should be considered as constituting physical and physiological normality. Here, as elsewhere in the human system, associated purposive acts in related muscular groupings,—as in the hands,—signify the adaptation of anatomic elements to subserve

purposes for which they are intended in some particular action-grouping. This is true even if the physiologic act be so faulty as to provoke an undue increase of some certain portion of the mechanism, thus not only continuing a physiologic error, but changing an anatomic relationship by which discomfort, pain, and disease may be produced.

## PART II.

## ETIOLOGY OF AMETROPIA AND HETEROPHORIA.

*The Eye in General.*—All of the studies in this article belong to the terrestrial eye,—the eye that refracts light from a rare into a dense medium. Lenses of rare material when placed in dense media act conversely to lenses of dense material that are placed in rare media. Based on this law, the dioptric systems of the eyes of the aquatic, the terrestrial, and the aerial animals are framed. For example, study of the microscopic sections of the various forms of the vertebrate eye shows, in the different phases of life-existence in this division of the animal kingdom, peculiarities of formation of each portion of the organ. The stage at which the existence of the ciliary muscle manifests itself, the varying shapes of the crystalline lens, and the varieties of curvature given to the cornea, all serve as illustrations of such differences.

As each subdivision of the animal kingdom evinces a diversity of ocular construction that is adapted to the wants of its possessor; and as each class of each division has a type of eye that is intended for some particular mode of life; and as each species of the same class exhibits dissimilarity in ocular construction: so man, in his many races and tribes, manifests characteristics that are related to his most persistent forms of livelihood.

*The Eye of Man.*—In man the converging bend of the ingoing ray is first made when the ray strikes the corneal surface and penetrates the membrane, the degree of refraction being dependent not only on the angle of the impinging ray and the medium through which it has come, but also on the tissues of the sagittal diameter of the eye through which the ray passes. Such an eyeball will allow retinal-image formation of extraneous objects with visual perception.

*Ametropic Eye.*—Alteration in the length of the antero-posterior diameter of the eye of man by which the rays do not cross one another properly is known as *axial ametropia.* Error in the curvature of the antero posterior surfaces of the refractive components is termed *meridional ametropia.* Change of refractive strength dependent on peculiarities in the substances of the dioptric media themselves is designated as *medial* or *index ametropia.*

The dynamic portion of the ocular apparatus is changing with its every physiologic act, and is subjected to each variation of local or general condition. For example, should the muscle-balance of the two eyes be set at variance from what is necessary for comfortable binocular vision by ametro-

pia or general disorder, such as neurasthenia, that moment every combined visual act becomes painful, and asthenopia appears. Again, when the accommodative act decreases by age or sickness, the muscle-action left may be employed to preserve proper vision for distant objects, and is unable to bring the reading point, for example, into the proper working distance.

It is the perversion of use that gives the abuse. It is not the business of a ciliary and an iris muscle to correct the focussing upon an infinite point. It is not the province of twelve extra-ocular muscles to set themselves into abnormal balance and strained relationship during every waking moment in the endeavor to bring about binocular fusion or fixation in a pair of antimetropic or anisometropic eyeballs. Muscle-rest and muscular equipoise are as necessary here as in other muscle-groupings, if not more so. Overtaxed and misused, the muscles exercise their evil influences on a series of delicate nerve forces which are almost constantly at work in their behalf; delicate, living machinery, like carefully-adjusted mechanisms, that apparently can work under almost every adverse circumstance, but when once out of order become, as a rule, uncertain and too often useless.

It is the elastic globe of the young, that is constantly used for near work, which suffers the greatest. Later, when the eyeball has attained its growth, particularly if the organ has been early and adequately protected, its tunics are firmer and its optical constants are more definitely fixed.

Undue stretching of the ocular tunics from muscle-disturbance is often as truly fraught with pathologic change as that which is consequent on uveal-tract inflammation itself. Ocular distention, with alteration of the dioptric standards and disturbance of the dynamic forces, gives rise to influences that serve both as a cause and as an effect.

*The Effect of the Eye on Mentality.*—The impairment of the functioning qualities of the ocular apparatus exercises its detrimental peculiarities on mentality. Perfect organs betoken good action, and good actions are productive of healthy mental development and growth.

Psychologic study shows that the mental horizon of any individual is in direct proportion to the peculiarities of the sensory channels through which its mental data are obtained. Mental capacity or quickness of mental grasp plays its part in the etiologic rôle more than is thought. How different from the intelligent mind of healthy childhood, culling every flower in the garden of knowledge without any effort, is the uncertain intellect of the dullard and the imbecile in their gropings after what is so laboriously gained! The one beautiful to watch in its development, its growth, and its activity; the other pitiful to observe in its slowness, its backwardness, and its many failings. The one using its organs like some smoothly working, undisturbed, and exquisitely balanced piece of machinery; the other employing its structures like some abused, disordered, and constantly harmed mechanism. In this latter, the eye, that most potential and yet most delicately contrived mechanism in the whole system, shows this ill use by definite change in structure. Here the increased physical labor to gain

a modicum of mental result is often bought at the price of disturbance of material that is disproportionate to the good that is gained. The acquired elongated globe of myopia, as so frequently seen in the so-called high-grade imbecile, is the prevalent form.

*General Causes of Ametropia.*—Increased solidarity is one of the greatest of etiologic factors in the production of ametropia. As previously said, the town-dwellers show a greater proclivity to organic change, of both inherited and acquired types, from functional abuse, than do their country cousins. From this, another great truth, a corollary of the former, stands out most vividly. The number of inhabitants of the cities of greatest commercial interests and of the most learning evidence a visual infirmity that outranks that of the dwellers in cities of less mechanical and mental importance. Comparison of the relative prevalence of high degrees and middle grades of compound hypermetropic astigmatism of the newer cities and sparsely populated districts of this country, with the number of cases of irritating, low grades of astigmatism in the large commercial centres, offers another example of proof.

As with cities, so with the parts thereof,—for example, in particular school-houses and in certain rooms of the same school. Persistence in near work, especially under adverse circumstances in such situations, tends to increase the percentage of gross ametropia and the number of individuals wearing lenticular and prismatic corrections in any one district. Yet it must not be forgotten that the presence of glasses on a number of individuals in any situation is not alone indicative of the greater prevalence of actually present asthenopia; because, in such cases, the disturbing factors are mostly set in abeyance.

*The Effect of Light on Ametropia.*—So, too, with illumination. As a rule, the sun's brilliancy is toned to a proper degree by the atmosphere. Indirect solar light is diffuse, and is different from intense artificial light, which, not to be injurious, should be filtered through appropriately tinted chimneys and shades, and rid of heat by the interposition of aqueous solutions of alum. The naked arc light and the exposed incandescent carbon loop and unshaded metallic ash-girdle, or mantle, are all too brilliant for employment during continuous near work. A translucent bulb covering the source of artificial illumination in such cases, while decreasing the brilliancy of the light, extends the field of lighting over such a great area that but a slight amount of luminosity is lost.

Inefficient illumination, either natural or artificial, constitutes one of the most important factors in the production of both the ametropic and heterophoric types of asthenopia. The ideal material is fluorescence, which gives light with but little or no heat, and the one that is being so widely experimented on to-day to render sufficient, steady, adequately diffuse, and practicable. For the present, however, it is necessary to be content with the various means for obtaining the best (not always the greatest) degree of incandescence from such materials as oil, gas, and electricity. This being

granted, it becomes desirable to know which of these is the best for any specific purpose.

A superior grade of petroleum or mineral oil, unaided by any of the various forms of zirconium, thorium, and yttrium mantles, etc., gives a less concentrated and a less powerful, though more comfortable, working light than ordinary illuminating gas. Gas-light increased in power by any form of incandescent ash, though weaker in intensity than the malodorous gaseous compound of the two atoms each of carbon and hydrogen known as ethine or acetylene, is, when properly diffused, much the more satisfactory and much the less harmful.

The unshaded loop of electrically incandescent fibre and the naked arc of free electric fluid are the most powerful of all practical light-givers, possessing qualities that, if not removed, may lessen their usefulness as illuminants, and give rise to conditions under which prolonged near work cannot be maintained.

In each of these light-givers, independent of the intensity of illumination, there reside peculiarities of quality that give rise to symptoms of asthenopia which render such forms of illuminants more or less objectionable. This is most probably dependent on the bleaching and the decomposing action of light at the extremities of the color spectrum on both rhodopsin and xanthopsin; this being especially noticeable in materials that are rich in cyanic blue tending to greenish yellow or white.

Irritating color-changes, exposure of the delicate and unaccustomed retinal mechanism to the ultra-violet rays and the thermic end of the spectrum, the want of proper diffusion of illumination from imperfect projectors, and the inequalities of focal concentration from incandescent points, all enter into the question of the production of asthenopia, and should be avoided as much as possible.

Granted that diffuse solar light, which can now be reflected into almost any desired situation by mirrors and prisms, is the least injurious of all illuminants, it may be stated that the best present form of artificial light-giver is probably any one of the many high-grade oil-lamps; provided that, after being placed in well-ventilated apartments, the light is properly positioned, is well diffused and correctly toned, and that it is made sufficient in intensity and rendered steady. These conditions can be obtained by experimentation only.

*Local Causes of Asthenopia.*—That astigmatism is one of the most potent factors in the production of asthenopia, there can be no doubt. Clinical study will evidence this, in spite of the attacks of unsupported theory. The minor degrees of the condition are those that furnish the greatest amount of asthenopia. Both functional and organic hyperphorias are more productive of asthenopia than either exophorias or esophorias of the same types, the latter conditions more frequently lessening and disappearing after correction of the vertical defects.

Ametropic asthenopia of overacting or even spastic type exerts its in-

fluence not only on the internal rectus muscles, but on all of the related muscle-groupings that are supplied by the oculo-motor nerve. For this reason, there is not only a convergence, but also an upward deviation of the eyeballs, as can be proved by phorometry. So, likewise, when the same character of asthenopia is continued until the ciliary muscle at last loses its hypertonicity and becomes secondarily paretic, the oculo-motor grouping becomes deficient in action, and the eyeballs are rendered not only exophoric, but also hypophoric by the antagonistic series; that is, in each eye there is an outward and downward tendency.

The variability between the efforts of convergence and accommodation explains why some cases of hypermetropia escape heterotropia.

Improper setting of frames, false positions of cylinder-corrections, dippings and planial inclinations of spherical lenses, and too strong prismatic effects, are all productive of eye-strain with physical change.

*Type-Writing as a Cause of Asthenopia.*—Another cause of eye-strain (generally of primary esophoric and later exophoric types) now becoming increasingly frequent in poorly nourished and improperly cared-for women of neurasthenic type, is the incessant labor imposed on ordinarily unused eye-muscles during the study of type-writing while watching the key-board of the machine before the necessary manual movements have become sufficiently automatic not to require any fixity of gaze upon the variously placed characters.

*Retinal Asthenopia.*—Though not strictly belonging to this article, and yet often referred to in it, there is said to be another asthenopic grouping which is dependent on disturbances that take place in the pigment layers of the retina, in which, in spite of proper lens estimation and the most careful hygiene and judicious therapy, there remains an imperfect visual functioning.

*Some Special Causes of Asthenopia.*—Various forms of asthenopia, especially of the retinal-fatigue type, may not infrequently arise from persistent gazing at badly-printed matter or print on highly-polished paper. Imperfectly formed script, poorly printed Arabic, Greek, Hebrew, and German type, diacritic points, peculiarities of each letter, such as the stems, the serifs, and the face, are all of the utmost importance as causative agents in the production of weak sight. So, too, with the size of written and printed characters. Small type of less than one and a half millimetres in height, badly constructed and fantastic letters, are among the worst of etiologic factors in the exposure of a latent ametropia and the production of a troublesome heterophoria. Distance and color of large sketching boards, tints of slates, and kinds of chalk used must also be considered. The want of double columns, the non employment of adequately leaded and interspaced matter, the selection of broad low-faced type, rather than the more easily recognized Gothic or high-faced type that necessitates less torsion, and the use of badly tinted and improperly calendered papers, all lend their aid to cause and production.

Faulty placing of the source of light in relation to the printed or written matter, persistent gazing at close or moving objects, and restrained positions of the body during the employment of the eyes add their weight. The same is true of occupations requiring much ocular near use, particularly those that necessitate continuous focussing and convergence on minute and closely situated objects. As a result of these habits and employments spastic tension of the ciliary muscle with forced convergence may take place. This, it is said, gives rise to disturbing actions on the extra-ocular muscles (especially the lateral recti, the inferior rectus, and the superior oblique), with consequent unequal elongation of the two globes and softening of the dural and the pial sheaths of the optic nerves at their bulbar entrances.

*Etiologic Factors in the Production of Heterophoric Disturbances.*—In the great majority of cases faulty muscle-equilibrium is the result of a transmitted over stimulation given to the ciliary muscle, the consequence of ametropia. In every hypermetropic eye necessitating undue use of the ciliary muscle, there is a disturbed relation of the extra-ocular muscle-groupings. It is a heterophoric asthenopia, which has its progenitor in an ametropic one; it is an associated error, the effect of individual imperfection. Though this be true, yet the variations of muscular power of the two eyes possessed by different individuals; the instability of degree of movement; the as yet unstudied, though determinate, innervation-impulses that are necessary for binocular equipoise of ametropic globes of definite size, curvature, and density; and the relationship existing between convergence, relative accommodation, and normal binocular focussing and fusion in all forms of ametropia, constitute some of the principal reasons why no two pairs of visual apparatuses (although subject to similar laws) can ever be placed absolutely under the same category of cause and effect. In the vast majority of cases muscle-disturbance is the result of unlike degrees of minor amounts of ametropia in two organs that are joined to work as one organ.

It is in the class of cases in which vision is kept to what is known as normal by an undue or an improper use of the ciliary muscle that the most disturbing heterophoric conditions with the most harmful general neuroses are found. Here the nerve and the muscle tensions are kept on a continual strain, which is a far different condition from that which is seen in gross ametropia, in which vision is so defective that it is almost impossible for the dynamic portion of the apparatus to continue to perform any useful correcting office.

The principal reason why the underacting and the paretic forms of heterophoric asthenopia (hypoexophorias, for example) are more frequently found in women is the want of out-of-door exercise, their proclivity to continued near work, and their greater neurotic tendencies. The depressing and the disturbing effects of toxæmia in the production of similar conditions in many such cases must not be forgotten. Related aural troubles, either

through associated intracranial fibres or by inflammatory reactions in contiguous tissue, must be considered also, especially in the overacting and the spastic types.

The finely balanced condition of the muscles from rapid development that is found at the age of adolescence may be, and probably is, one of the reasons why the ocular groupings of muscles which are so delicate suffer so greatly and so early in ametropic conditions at this period of life. To illustrate their delicacy of equipoise, they express their cessation of reactions the earliest in natural conditions, such as dreamless sleep with its neuronic rest; in induced states, such as hypnotism with its incomplete and unremembered neuronic acts; in hysteria, in which the neurons are disturbed and ofttimes rendered temporarily inactive, and in many of the most diverse forms of pathologic conditions of gross organic nerve disease.

In addition, there often seems to be a general neurosis at hand. In these instances the unstable muscle-equilibrium apparently not only acts as an exposer of greater local disorder, but also serves as a precipitant, as it were, of a general attack, or as a constant exciter, until the general condition becomes measurably impaired.

The cephalalgia found in these cases and seen especially in women of the neurasthenic type is, as a rule, situated in the occipital, the nuchal, and the cervical regions, extending in some instances as far down as the dorsal areas. Confusion and nausea that are increased by gazing at rapidly moving objects, or are induced by repeated change of focus from distant to near objects, with inability for continued mental application, are, amid the general expressions of petulance, irritability, etc., both symptomatic and etiologic factors. As illustrative, megrimic subjects, who are ordinarily neurotic in type, unless relieved by the employment of lenses to correct an existent ametropia which removes the tension on the exterior and the interior ocular muscle-groupings, are prone to have their attacks lessen at about forty-five years of age,—at the period when muscle-action becomes lessened; thus showing that heterophoric disturbances have much to do with the production of such conditions. Moreover, as confirmatory, the condition generally makes its appearance at the time when the eyes are begun to be used more or less continuously for near work.

This can be better understood when it is considered how many of the intracranial nerve-trunks attach themselves to both the motor and the sensory portions of the two ocular bulbs; and that there are an incalculable number of association-fibres brought into play during the interfunctioning of the two organs. Besides, the two retinal expanses have their embryologic and anatomic counterparts in the cerebral sight-centres in the occipital cortex. Adequate knowledge of these structures—that are highly vascular in character; that are linked by plexus, nerve, ganglion, and neuron; and that perform their highest types of combined sensory and motor actions—soon makes it evident that faulty focus with imperfect impression and disturbed harmony of dual action in the two end-organs (the

eyes) may not only act as precursors, but also serve to give origin to some of the most common functional and organic neural disorders.

It is not, however, always the mere presence of the ametropia that gives rise to asthenopia: heterophoria can exist with emmetropia. In great measure the interrelation of the two organs, the character of occupation, and the state of the general health are often the fundamental conditions upon which eye-strain is based. Ametropia may exist and remain latent and undisturbing, provided that the above factors are never brought into existence. When it becomes disturbing, the resultant condition—asthenopia—is simply an expression of tire, which, when continued, may give rise to secondary changes in and around the ocular bulbs: changes that at first are often spastic and hyperæsthetic in character, but which sooner or later terminate in pareses and anæsthesias. For example, the symptom-grouping of heterophoric asthenopia of paretic type in which there is a transitory functional exophoria is, during near work, associated with headache and ocular disturbances that may be either causative or resultant of indirectly induced hyper-convergence and undue accommodative acts. Extending in regular lines along reflex routes, the interplay between the loss of general tone and the local disorder soon appears, and the visual apparatus is rendered irritable, uncomfortable, and at times useless for its accustomed work. Moreover, the ametropic fault, with its heterophoric result of organic type, be it "structural," "insertional," or "innervational," if slight, may, while giving an apparently normal physiologic act, and thus escaping recognition by the careless or the ignorant, become more harmful, on account of its being hidden, than a gross error, which, unable adequately to correct itself even by extraordinary tension and false response, exercises less perverted action and injurious eye-strain.

*General and Special Peculiarities of Ametropia and Heterophoria.*—Unfortunately, this is an age of prolonged undue convergence, a condition that consists in a dropping of the visual plane with accommodative action and fixation on close medially placed objects: all factors of the most disturbing functioning type.

The movements of the two eyeballs during binocular vision, as ordinarily pursued, are far more complex than is generally taken for granted. Not only is there an increased tension in all manner of changeable proportions given to the muscles that are affected directly in any given combined movement, but there are a number of indirect relaxations of the series of antagonists. The conditions are variable during the relative positions of the two, ofttimes unequally sized and shaped globes; eyeballs that are working to different amounts of impulse and grades of energy during the constantly varying states of the general health. It is extremely rare that there is an increase of refraction in a single eye that is dependent on ordinary eye-strain, thus offering an additional reason why in binocular myopia, monocular vision is often better for the comfort of the patient and the safety of the functioning visual organ.

Although there are geometric standards from which all variations are to be made (the positions assumed during physiologic rest probably representing the primary position from which all motion should be gauged), and which should be sought for in every case, yet, just as with the eyeballs themselves, a practical condition for normal and healthful action must be considered as the more desirable rather than the ideal one which may frequently simply signify harm.

Ametropic asthenopia, expressing itself as a recurrent inability to sustain any fixed focussing point, is a frequently complained-of subjective sign of hypermetropia. As explained later, it expresses itself most prominently as an intermittent weakness of the ciliary muscle which is being over-used to sustain distant vision, thus leaving an inefficient amount of focussing power for near work.

There can be no doubt that the reason ascribed by Donders and his predecessors, that some cases of asthenopia are overcome by an "active convergent strabismus," is true. The continuance of the apparent undue spastic or active convergence, however, beyond middle life, in which little or no power of accommodation is left, is dependent in great part upon hypertrophy of the exterior ocular muscle-series that produces esotropia. The fact that some hypermetropes converge during sleep is also corroborative. In addition to these reasons, such peculiarities as the size of the angle *alpha*, the length of the base line, etc., must be considered as having weight in the production of heterotropia. If with these the innervational relation which enters into every case be studied—for example, the one which exists between convergence and relative accommodation—it will be established that for every finite binocular point an undue convergence is made, and that the unnecessary combined inward movement, in spite of the ordinary fusional effort, will be continued even after all accommodative effort is removed. In fact, it will, in nearly every case, make its appearance as soon as the innervational impulses come into existence.

As regards the spontaneous disappearance of esotropia, either the refractive constants have become so increased in quantity or density as to do away in part or entirely with the necessity for dynamic correction before hypertrophic muscular tissue material has been obtained, or, as is seen in the foreign and the older contingent of the large public ophthalmic services here in the United States of America, the unscientific correction of ametropia (especially of astigmatism) has led to an amblyopia exanopsia of the greater deviating organ with an apparent bulbar parallelism.

*Relation of Vision to Heterophoria and Ametropia.*—It must not be forgotten that comfortable vision may mean the existence of an actual heterophoria, both manifest and latent, as may be determined by any of the series of refined muscle-testings at hand—degrees and types of muscular error that to the geometric muscle-cutter are abominations, in spite of the fact that the exterior and the interior series of ocular muscles are relational and exist only as a part of an immense and unstable motor apparatus. It

should also be understood that the condition may be the result of either an increased tension of certain muscle-groupings or an impaired action of the opponents: one, a positive or a spastic condition; the other, a negative or a paretic state. Again, the fault not only may be organic in character, in which the false equilibrium persists even during want of muscle-action, as in sleep, but it may also be functional (and most frequently is), as, for example, when dependent on ametropia, the faulty equilibrium disappears the moment that the functioning power of the combined two eyes becomes properly regulated.

By reason that seldom, if ever, any two paired eyes are exactly alike in their refractive constituents and hence in their motor impulses, every such pair of eyes must have its two individual emmetropic models aimed at in different ways, and, as a consequence, unequal innervations must be given to the two sets of focussing and governing muscles.

Orthophoria, therefore, may be the result of an anomalous balance, at best, and the normal relational equipoise may constitute a different innervational factor in each of the two organs. Moreover, the visual apparatus holding a part of the vascular and neural channels of the system, any peculiarity of structure by which imperfect optic (and hence incorrect impressional) results are obtained must not only disturb the special apparatus itself, but also set a train of injurious symptoms into play, by which the most diverse and the most peculiar inequalities of action become established. In this category may, with some show of scientific propriety, be included so-termed reflex epilepsy, or that which depends on some peripheral cause and which is amenable to the removal of the source of irritation.

On the other hand, the intimate connection of each organ in the organism during physiologic activity is lost sight of by those who, either from ignorance or caprice, see the initial lesion of every series of bodily complaints in the organ of their own special work, and thus fail to realize the ever-harmonious though, to them, frequently conflicting groups of symptom-complex. Just as it is true that ametropia and heterophoria can and do produce impaired health, so, conversely, will impairment of health give rise to heterophoria and exposure of ametropia. In other words, the refractive and the muscular errors constitute a leak through which valuable nervous energy is lost, allowing irregularity of physiologic action with the production of faulty physical change, which in turn acts as a causal agent for secondary disorders and harmful functioning,—an injurious interplay of cause and effect. Here is the major secret of the fallacy of the current belief that ametropia is simply a defective optical condition, to be remedied by the selection of some apparently useful, though quite frequently injurious, combination of lenses. Overtaxed neurasthenics, unexercised high-grade neurotics, and subjects with but little physical strength whose powers of convergence and accommodation are set awry by unscientifically sought-for lenticular adjustments (that may even give the best visual acuity in each individual eye), may frequently have their ocular movements and impulses

so disturbed as to render the corrections that have been given not only troublesome and, unfortunately, unbearable, but actually harmful to the patient's general health.

*Changes in Ametropia and Heterophoria.*—So-called regressive hypermetropia and progressive myopia are, as a rule, simply refractive signs of the grade of bulbar distention. In most cases the very causes that are producing an increasing myopia have been the ones that have diminished a previous hypermetropia. A stretching hypermetropic eyeball may, though of course rarely, pass through more stages of irritation and inflammatory change without the refraction error ever reaching the myopic standard than a well-made congenitally myopic one.

It is a mistake to assert that an ametropic eyeball of progressive type does not exhibit any signs of pathologic change,—"that it is healthy and that it is normal." The imperfect vision and the faulty correlation between the two organs show at once a physiologic error that must be an evidence of abnormal physical change.

Early in the development and growth of the child, the normal eye—the one of short axial length—grows somewhat more spherical. Its refractive media change their indices, and its altered shape becomes more or less fixed. This is gradually accomplished to varying degrees early in life. Should this take place equally up to a definite amount that approaches emmetropia as nearly as possible, it will be found that the ocular tissues are firmer, the extra-ocular muscles exert less compression, and the intra-ocular fluids produce less distention than they would had the work of development and growth been improperly performed in the two organs. Towards the fourth and the sixth years of life, especially in countries of advanced civilization and fine handicraft, unusual activity is given to the two organs. At this period, or even before any future expressions of inadequacy and inequality in the two eyes begin to manifest themselves, until at varying later periods, more particularly about that of the time of adolescence, the processes pass beyond what may be considered to be of no harm, and thus give birth, as it were, to the first expression of both ametropic and heterophoric asthenopia; at this point, eye-strain expresses its ordinarily recognized signs.

At what point this distinction can be made is uncertain. No optical law can be obtained to govern it; no geometric formula can be expressed to solve it; and no minor degree of expression of pathologic condition can be assumed as normal in any particular case. It is a question of relationship of condition of structure and the employment of individual organs. What may be necessary to produce a coarse pathologic picture in one eye may fail to exhibit but the ordinarily used condition of so-called healthy working tissues in another. What may be the exponent of practical health in one organ may be the representative of disease in another.

*Local Causes of Hypermetropia.*—Accepting a hypermetropia of about three diopters' strength as the refractive expression of the human eyeball at birth, as a rule, about one-third or one-half of this amount is lost, not only

by further normal development of the eye, but in many cases by pathologic change.

To effect this change of supposedly fixed ocular "constants," many factors in addition to the ordinary congenital abnormality of too weak a dioptric apparatus are said to be brought into play. Augmentation of the retrobulbar contents of the orbit, from vascular change, excessive adipose tissue, neoplastic formation, etc., particularly when situated in the extra-ocular muscle funnel, by pressure have been assumed so to shorten the antero-posterior diameter of even an emmetropic or a myopic globe as to produce a marked hypermetropia. Forward displacement of that portion of the retina which is situated in the macular region and which remains functioning, by localized chorioidal disturbance or submacular exudate, may so lessen the power of the refractive constants in the visual axis, as to either reduce a myopia or an emmetropia to a hypermetropia or increase an already existing hypermetropia.

Alterations in the anterior pole of the eyeball, such as inflammatory, traumatic, and post-operative flattening of the cornea, etc., not only are accused of giving rise to meridional astigmatism but are said to produce hypermetropia. Lid-pressure on the anterior face of the eyeball may also, it is stated, produce both the axial and the meridional forms of the defect.

Aphakia (a medial defect) is one of the most frequent causes of acquired hypermetropia. It produces a lower index of refraction of the dioptric media (with either a resultant increase of a previous hypermetropic error, a change from a myopia into a hypermetropia, or a decrease of a myopia) than that which existed before the appearance of the condition.

*Dynamic Exposure of Hypermetropia.*—The dynamic exposure of hypermetropia from local disturbances or general disorder has nothing to do with this question. Diphtheritic, artificial, toxic, and intracranial paralyses of accommodation, all may mean the removal of supplementary lenticular strengths to inefficient—hypermetropic—eyeballs, by which the refractive error had been hidden. Lenticular and ciliary muscle disturbances from senility or glaucomatic processes are also frequent causes for bringing into functional existence, as it were, a hypermetropia which had previously remained concealed and undisturbing.

If unprotected, many such cases gradually pass in their refractive equivalents from a state of hypermetropic astigmatism to a myopic one. In these types emmetropia is never seen. The change is a pathologic one.

*Emmetropia, as Practically Seen.*—The optical equivalent of emmetropia is but a theoretic standard born of the lecturer on optics, the statistician, and the mathematician. The very fact of the eye's being emmetropic in its refraction is proof, almost without exception, that it is at a temporarily fixed point in its passage from a hypermetropia to a myopia—the result of a stretching process that can be accomplished only by an inflammatory condition, no matter whether it is observable to the ordinary methods of precision of to-day or not. Likewise with the exterior ocular muscle-

groupings, morbid changes of a related type must also be resident in them; changes that require the greatest precision to determine.

*Significance of Terms Used in Ametropia.*—By reason of constant usage the absurdity of the terminology now employed—"hypermetropia," "myopia," and "astigmatism"—is not apparent until it is applied to some other sense-organ; for example, the auditory apparatus. Why should not "hypermetauria" be the term given to an auditory organ so shaped from a standard known as "emmetauria" as to permit the ability of hearing only distant sounds, and some relevant Greek or Roman coinage be offered to express a forced inclination of the head to one side to hear better at a distance (just as myopia signifies a nipping together of the lids to see better at a distance)? and if imperfect sound focus on the sentient tips of the auditory apparatus be determinable, why not apply "astigmatism" to that condition? In truth, the constant application of the terms "hypermetropia," "myopia," and "astigmatism" has robbed them of their meaninglessness.

As usual, in these studies as elsewhere, it may be found that the earliest theories in regard to physiologic optics are the best, being untrammelled with differences of opinion that too frequently are but mere attempts at perversion and nonsensical dissimilarity. As early as the middle of the sixteenth century Maurolycus gave a most accurate account of both "shortsightedness" and "long-sightedness." In the former, he says, "the pencils of rays converge too fast, and come to a focus before they reach the retina." In the latter, he writes, "those pencils do not converge fast enough, so that the foci are beyond the retina." He further gives the correct reasons why these optical defects are relieved by concave and convex lenses.

*Prevalence of Grades of Ametropia.*—In this part of the world (the eastern seaboard of the United States of America), low degrees of H + Ah passing into Ah, signifying a less progression of the ametropic stretch than formerly, are, when properly sought for, found to be quite prevalent. This is followed by Ah, then H or Ahm and Amh, M + Am (fortunately rapidly lessening), and, lastly, H. The prevalent axis of astigmatism in H + Ah is 90°; that at 180°, which is designated as "against the rule," being particularly seen among the educational and commercial types of the Semitic race. Unsymmetric axes are often found. The two principal meridians frequently exhibit a relational degree of angulation, though the relative differences need not necessarily be placed, as some have attempted to show, at five, ten, fifteen, and twenty degrees each, which are simply expressive of the coarse numeration on the ordinary trial-frames. In fact, less differences and more unequal ones can be easily demonstrated by repeated work with adaptations to optometers and test-frames, by which finer differentiations of cylindric lens-axis can be made.

As should be expected, the unsymmetric axes are most common in the minor degrees of ametropia, being particularly noticeable in Ahm, Am, and M + Am. It is also certain that in many cases the angle of the astigmatic meridian that is used is not the same in monocular as in binocular fixation.

The percentage of the amount of astigmatism is somewhat in excess in the female subject, particularly in the young of dolichocephalic type. The relative proportion of defect that is situated "against the rule" is proportionately greater in the eye of the myope than it is in the eye of the hypermetrope, this being in a measure accounted for when it is realized that the latter condition is ordinarily a congenital one and the former nearly always a pathologically acquired one. Oblique positions of the principal meridians of astigmatism, particularly when the curvatures are unlike in degree, are more prone to give rise to asthenopia than are the vertical or the horizontal ones.

Hypermetropic astigmatism "according to the rule" or "with the rule" is not so disturbing to vision as that which is "contrary to the rule" or "against the rule."

Compound myopic astigmatism in the rising native-born generation in this part of the United States of America is becoming rarer. This is dependent on the halting, as it were, of progressive ametropia by carefully chosen and properly fitting corrections for H + Ah, the betterment of environment, the more judicious use of the eyes, and the greater tendency of the age to out-of-door sports and exercise.

Heterophoria is relatively more common here in the United States of America by reason of the rapid ingestion of improperly prepared foods, greater mental and physical activity with less personal care, as well as climatic influences which are most enervating in character.

As with the individual, so with the masses. For example, the English-speaking nation constantly gains a height in mechanics, physics, science, etc., that can never be reached through the same easy flights by the German. It is the close application for detail and the great reverence for the work of the past in the Teutonic mind that are balanced against the intuitive reaching out for the new and the ignoring of the old of the English mental make-up. New fields for work, new countries to be opened, with new ideas to be brought into play, for the American and the Briton, have all set aside the traditions of the past and planted the spirit of enterprise into the brain and the brawn of the English-speaking peoples, and have thus carried them beyond the shop, the mill, and the factory.

It is the plodding nation in which myopia is the most prevalent,—the nation of human automatism, as it were. It is in those nations in which individual hygiene is at a minimum, in which life is sedentary, and in which the tissues are relaxed and possess but little resistance, that the percentage of gross ametropia is much the greater.

At present the eye is used much more than formerly. Life-work is commenced too early, and the necessary preparation is more quickly accomplished, though with greater wear on the involved tissues. In this connection, nevertheless, the apparent increase in the number of young subjects who find it necessary to use correcting lenses means not only that modern civilization is more exacting on physical structure, but that medicine has

recognized the value of the artificial lens in removing ocular and general symptoms which previous to the advent of the ordinary correcting of ametropia remained unbenefited, except temporarily by bloodletting, emesis, purgation, etc.

*Local Causes of Myopia.*—Any diminution of the density or of the tonicity of the tissues in the retrobulbar portion of the orbital cavity, especially when situated in the exterior ocular muscle funnel, is said to give greater opportunity, particularly in the weak and the neurasthenic, for injurious laterally situated pressure-processes to exert their influences for the production of increasing axial myopia.

Retinal detachment situated in the focussing area of the eyeball in which functioning remains will reduce a myopia. Chorio-retinal changes in the macular region, however, no matter how minute they may be, and how slightly visible they are to the ophthalmoscopic observer, have a bearing on the proper functioning of the parts. Asthenopia, though not necessarily present in all such cases, is the occasional accompaniment of a consequent imperfection of retinal impulses.

Whether the hypertrophied portions of the ciliary muscle of hypermetropia have any effect on the compression of the globe in its ante-equatorial zone, and thus indirectly induce an increase in the axial length of the eyeball with a tendency towards the production of myopia, is still uncertain. By some it is asserted that an abnormal pressure of the superior oblique muscle on the eyeball, with increased venous congestion, is prone to produce the condition. In regard to this statement, it may be said that, although the two oblique muscles tend to remove the eyeballs from the position of undue convergence, yet the extent of length over which the inferior oblique muscles press against the eyeballs, and the dragging of the insertion of the superior oblique muscles on the edges of the optic nerves, more than compensate the good that is obtained by the divergence.

Abnormalities of the retrobulbar portion of the optic nerve itself, not permitting proper movement of the eyeball, have been spoken of as exciting causes. Peculiarities of scleral structure, particularly of those portions that are situated in the position of the optic-nerve sheaths, by which a disposition to an undue stretching of the ocular coats is induced, have been assumed as casual factors. To others idiosyncrasies in extra-ocular muscle situation, by which the venæ vorticosæ are unduly pressed upon, have seemed of sufficient influence to give rise to the condition. Irregularities in the orbital walls and borders, by which false movements of the ocular globes are obtained, appear to some investigators to be among the chief casual elements at work.

That there is a tendency to heredity in the form of the myopic eyeball, just as there is in any other end-organ or in any facial peculiarity, there can be no doubt.

In the dynamic form of cause in which there is ciliary spasm with too great focussing power of the eyes, the constantly employed focus of vision

is too close, giving rise to a persistently undue convergence which produces, as one of its results, an increased curvilinear pressure on the temporal side of the globe by the external rectus muscle, thus tending permanently to increase the antero-posterior axis of the ocular globe.

Though the average time of life at which the ametropia changes from hypermetropia to myopia is during early adolescence, when final development is the most rapid and at its greatest, yet the stretching of the eyeballs, which manifested itself as a lessening hypermetropia in such cases, has been in existence for several years' time. It is an error, therefore, to assert that such a hypermetropic eyeball has not exhibited any pathologic change: a change that is manifest with both the ophthalmoscope and the microscope.

If the productive causes should come into existence during later life, the ocular changes would not only tend to give a similar optical result, but also produce peculiarities that would be more greatly detrimental to the integrity of the organ itself. This is shown, for example, by the malignant forms of the affection, which are generally of high-grade type, and which are found among such subjects as sailors and laborers whose eyes are never persistently applied for near work: a mere symptomatic expression of a coarse local disease. In such cases a sedentary life and a weakened constitution, even among the illiterate and unlearned, add their injurious influences to the local changes. This is clearly seen in young subjects of the cardio-vascular types, in rheumatics, and among those who are disposed to lymph-stasis. Formerly, in the malignant type of the affection, straining at stool and constipation were noted as immediate precursors of attacks of ocular congestion, hemorrhages into the vitreous humor, and retinal detachment.

Generally, in such cases, when both of the progenitors are myopic, the organ is born into the world with its greatest liability to physical elongation. Less liable when only the mother is affected, and with the least tendency when the condition is present alone in the father, it seems to be dependent more on the maternal ancestry than on the paternal.

The congenital inflammatory types, which are becoming comparatively rare, are, as a rule, simply optical expressions of actual disease that has been caused by intra-uterine change, which at times may be superadded to a similar hereditary taint. At times there may be a series of hereditary tendencies of weak and unresistant tissue which, when subjected to ordinary amounts of wear and tear, express themselves by distention and compression-processes that increase the equivalent of the dioptric apparatus of the organ.

In every instance such processes must be considered as the diseases, and the optical expression—the so-called "myopia," for example—must be regarded as but one of the symptoms. In some of these cases almost imperceptible changes may occur which in many instances are so slight that repeated examinations are necessary before any visible expressions of

progression of the disease can be recognized. In others periodic outbreaks of inflammatory change, with fleeting gross signs and resultant cicatricial physical change, constitute the main characteristics. Constant in type and rapidly leading to destructive blindness, each class comprises in itself a special variety of the generic form.

The inflammatory processes may have their origin at almost any point in the ocular bulb. Nebulæ from previous corneal inflammation—necessitating too close an approximation of near objects in order to enlarge the size of the retinal images, and thus giving rise to too strong convergence and spasm of accommodation, with their train of distention-symptoms—may start the process. Trauma, with secondary changes in the crystalline lens; ciliary spasm, with muscle-irritation and congestion of the uveal tract; gross anatomical peculiarities, and undue stooping, with passive hyperæmia by pressure on the cervical veins, all play their etiologic *rôles*. Increase of corneal curvature, increased density of the constituents of the dioptric media, forward displacement of all or of a portion of the crystalline lens, and backward ectasias at the posterior pole of the eye, are said to constitute the main grouping of factors in the production of the condition.

The constant employment of the ciliary muscle to keep the lenticular mass in a surcharged state, and thus allow the crystalline lens to serve as a stronger condenser, in an acting eye that naturally has too little focussing power, may at times cause the muscle to be spasmodically affected. During binocular vision, with the requisite undue convergence, and also in near work, when greater action is essential and increased congestion is obtained, is this especially found. Thus is frequently produced a symptomatic myopia and sometimes a decreased hypermetropia, which, particularly in young subjects, may become so fixed as to render distant objects very indistinct to patients belonging to the former class. Often this condition is too truly but the initiative for disturbing irritation and inflammatory signs.

The symptoms of the transitory forms of lowered hypermetropia and increased myopia, the results of diabetes mellitus, are, as a rule, unassociated with gross inflammatory ocular change. The temporary varieties found in plastic iritis and irido-cyclitis, and which are of much lower grade than the former, evidence a greater degree of meridional inequality than the lymphatic types.

Spasm of accommodation, with its associated irregularities of action between the extra-ocular and the intra-ocular muscle-groupings, which is often produced by prolonged work at some near point, is apt to bring about harmful uveal and retinal changes with antero-posterior stretching of the eyeball.

Again, the ciliary muscle, in consequence of an improper action from disturbed extra-ocular muscle-balance, or as the result of inflammatory congestion and exudate (so often seen in keratitis, scleritis, uveitis, retinitis, and neuritis), may exert a more or less fixed influence on the lenticular

mass, allowing it to assume too gross a shape and thus give the eyeball an increased refractive power.

An increase in the refractive strength of the crystalline lens from sclerosing processes is found to give rise to a temporary form of myopia which is known, by reason of a non-necessity for the continuance of the use of condensing lenses during near work, as "second sight."

*General Causes of Myopia.*—Nationality is said to play its part in the production of myopia. The reason that some countries—for instance, Germany—have had so much myopia is on account of the past want of proper correction of hypermetropia and hypermetropic astigmatism, before myopia became a national defect, which now, happily, is fast decreasing as the result of scientific investigation and sanitary regulation. In this part of the world the condition has, in quite a number of adult cases of the present day, been caught in the very beginning stage of the error,—mixed astigmatism.

The prevalence of the eyeball that had stretched sufficiently to give a gross myopia was greater here upon the eastern seaboard of the United States of America a quarter of a century ago than it is at the present time. Not relatively, by reason of the greater number of asthenopic hypermetropes that are being corrected, but actually so,—fewer cases being seen. This is, no doubt, dependent on the increased number of eyes with minor degrees of hypermetropia and astigmatism that have been corrected and thus prevented from passing into the myopic state of refraction.

By some it is said that the condition is unknown among such people as the Nubians, the Laplanders, and the Patagonians. Some authorities assert that it is not seen in the peasant classes of Europe. In this portion of the United States of America, since slavery has been abolished and higher education has been given to the various grades of Negroes, myopia, independent of that which is produced by gross ocular inflammation, is not infrequently seen in the advanced student classes of this race.

*Theories in Regard to the Etiology of Myopia.*—Contrary to the findings of some investigators, that the vertical diameter of the orbit of the myope is relatively increased, it is asserted by others that in low orbits there is such a variation in the course and the insertion of the superior oblique muscles as to induce compression of the eyeball with the production of myopia. This theory of the compressing effect of these muscles on the eyeball is understood when their insertions and origins, as well as the directions of their mechanical forces, are considered.

This combination of muscle-pressure (especially the superior oblique muscles in downward convergence, and their dragging effect on the lamina cribrosa at the optic-nerve entrances, with the harmful influences of occupations that necessitate undue vertical movements of the globes) constitutes a part of the mechanics in the stretching and irritative and the inflammatory and degenerative processes that are so often seen in this condition. Compression of the vorticose veins by the inferior oblique and the external rec-

tus muscles has also been assumed as an indirect factor. No matter whether myopia be an anomaly or an acquired malformation, the condition frequently sets a whole chain of circumstances at work to force the posterior pole of the eyeball into the free funnel-like space that is situated between the bellies of the posterior portions of the four straight extra-ocular muscles.

How much dependence can be placed on the theory that a hyper-inclusion of mesoblastic vitreous material into the secondary optic vesicle is the causative factor of myopia is uncertain, although the suggestion of such an exaggeration of mesodermic inclusion has a number of points in its favor. Wasting diseases as found in infancy serve as most important and not thoroughly appreciated factors in the production of the disorder.

As the stretching process of axial myopia is especially marked at the posterior pole of the eyeball, the constituents of the vitreous humor must be separated, and thus the refractive index of this dioptric medium must be slightly lessened. The increase in the length of the eyeball, however, is relatively of so much more importance in the ametropic change that the decreased density of the vitreous humor practically amounts to but very little.

The assertion that the improperly termed posterior staphyloma of Scarpa (which is not a bunch of grape-like series of ectasias) is a necessary accompaniment of the majority of highly myopic eyes is, in the light of recent investigation, very uncertain. Care should be taken to compare the condition with what is known as "conus." Determined as early as 1801, and associated with myopia by Arlt over fifty years later, it is said by some to be congenital and to occur, as a rule, in the position of the ocular cleft. It is assumed to be different from the stretching and tearing of the ocular tunics that are found as the results of acquired pathologic distention: conditions which must, according to some, cause the greatest reactions and atrophies to appear at the temporal borders of the optic nerve-head in low and medium degrees of myopia, and later at the nasal edges of the optic disk in the higher grades of the same form of refraction-error.

Truly, myopia cannot, in such a light, be considered as an adaptation of an organ to the functions of a superior race. In most cases it is a pathologic process with a dioptric equivalent of near-sight.

*Causes of Astigmatism.*—Traction on the cornea, as seen in peripherally situated cicatrization-wounds, either accidentally or purposively made, superficial tumor-growths at the corneal limbus, symblepharon, and pterygia, all may be productive of astigmatism. Direct and indirect pressure on the cornea or the sclerotic, as, for example, from new growths that are situated either in the orbit or in the eyelids, and spasmodic contraction of the orbicularis muscle, are said to give rise to corneal astigmatism. In contradistinction to this, irregularities of the tunics at the posterior pole of the eye may produce an astigmatism by faulty curvatures of that portion of the retinal sheet which is situated in the macular region.

Astigmatism is also said to be caused by intra-ocular pressure and tonic

contraction of the exterior ocular muscle-groupings. The variety that is produced by lid-pressure is either a temporary form of the regular type, or, more frequently, an irregular one of meridional character. In this connection, it must not be forgotten that a spastic condition of a series of extra-ocular muscles, as is occasionally seen in some types of nerve disease, may give rise to temporary forms of the condition.

In the causation of astigmatism both surfaces of the cornea and of the lens, as well as the density of the different portions of the two masses (especially in the course of the principal axial lines, thus making four surfaces and two thicknesses), must be taken into consideration: likewise with the aqueous and the vitreous humors. For example, in saccharine diabetes it is not inconceivable that there may be not only an ordinary ametropic change brought into existence but an astigmatic one as well.

Astigmatic errors from differences in the principal meridians of corneal curvature can be changed wholly or in part by relative irregularities in lenticular curvatures through unequal ciliary-muscle action.

Astigmatism, especially that which is said to be "against the rule," may be produced by differences in the curvatures of the anterior and of the posterior surfaces of the cornea alone. Fixed lenticular astigmatism is at times dependent on an oblique position of the crystalline lens as one of the results of traumatism, though it has not infrequently been observed as a condition of congenital origin. The acquired form of astigmatism that is seen after corneal section is almost always produced by a flattening of the corneal membrane in the meridian that is situated at right angles to the middle of the circumlinear section of the cornea, with a decrease of the radius of curvature in the corneal meridian that is parallel with the incision. When marked, it is generally dependent on an improper approximation of the lips of the wound, the edge of the stump usually being shifted in front of the flap,—a difference of twelve one-hundredths of a millimetre and thirty one-hundredths of a millimetre, giving, refractively, an alteration of two to five and a half diopters; this in a measure being increased by extra-ocular muscle-action.

As is well known, not only can increased spherical power and heterophoric action be given to an eye, but astigmatic effects can be obtained by spherical lenses that are placed obliquely or are tilted before the eyes, thus bringing new optical factors into existence. At times, improperly corrected subjects have obtained more efficient or even proper artificial correction of astigmatism by bending the planes of their inadequate lenses before their eyes.

*Some Peculiarities Seen in Astigmatism.*—When the degrees and the principal meridians of astigmatism of each eye have been determined separately, it will at times be found not only that the amounts of the defect change when the examinations are made with the two eyes simultaneously, but that the relative angles of the two principal meridians likewise change. This is possibly to be accounted for in a measure by the

action of the extra-ocular muscles in producing, both directly and indirectly, different degrees of tension on the eyeballs during their combined functioning: a newly-balanced equilibrium of the muscles, as it were, with a peculiarity of obliquity of position of the two eyeballs themselves.

It is a curious fact that quite a considerable number of cases of simulated blindness may be directly traced to uncorrected medium and high grade astigmatism.

In opposition to the supposed correcting power of depth of penetration upon astigmatism during vision through a microscope, astigmatic eyes are disturbed in their use of the instrument, while the value of their visual work is lessened.

*Production of Presbyopia.*—The age at which the recession of the near-point begins depends on the refraction of the eyes and the condition and the occupation of the individual. The child that is eleven years old has not the same closeness of near-point that he had when he was seven years of age. The athlete who never uses his eyes for close work has the recession of his near-point postponed to a later period than the sedentary neurasthenic whose life is spent amid his books; while the middle-grade myope, constantly employing his uncorrected far-point for near work, never can be made to appreciate his accommodative defect, even though all of his focussing power be lost and his near-point and far-point stand as one.

*Effects of Solanacea in the Production of Asthenopia.*—Ametropic asthenopia may be made more apparent or may even be brought into existence by the internal administration or the external application of weak doses of one or more of the many forms of solanaceæ.

## PART III.

### SYMPTOMS.

"Ametropia," a symptomatic term, is a dioptric expression of an abnormal state of refraction. Clinically, it is divided into three groupings: hypermetropia, myopia, and astigmatism. Considering their symptoms in order, the following subdivisions are obtained: A, Local objective signs; B, General objective signs; C, Local subjective signs; and D, General subjective signs.

#### HYPERMETROPIA.

In this type of ametropia, in which the eyeball does not possess sufficient static focussing power, the symptoms are plainly, and frequently painfully, manifest.

A. **Local Objective Signs.**—The eyeball, which, as a rule, is situated to the temporal side in the orbital cavity, is small in size, particularly in the diameter that corresponds with its antero-posterior axis. The scleral coat is flattened in its anterior portion, and is bulging and abruptly curved in the equatorial region. In the axial form of the error, the angle *alpha*

is wide, becoming so broad in some high-grade cases that the point of penetration of the line of vision on the anterior surface of the cornea may be situated so far to the nasal side as to produce an opening angle that is, at times, almost double the width of that which is found in emmetropia. At the posterior pole of the eye, the fovea centralis is situated quite far temporally from the outer edge of the optic nerve-head. The position of the centre of motion, or the centre of rotation as it is sometimes called, is shallow in such eyes, though it is placed relatively near to the retinal plane. In the majority of cases, there is an apparent exotropia of slight degree, and the eyeballs are freely mobile.

Esotropia which makes its appearance quite early in life, although not so common now in this community as formerly, is an evident symptom of the middle and the rather high grade hypermetrope. Periodic and noncomitant in type, and in direct association with the amount of accommodation-effort at first, it soon becomes alternate in character, until later, if not corrected, it passes on to the comitant, constant, and amblyopic variety.

Nearly every grade of hypermetropia manifests its presence by ocular-muscle disturbance. Impairment of the visual functions of the weaker organs is, as a rule, found in the higher degrees of the error, while the minor degrees of the condition produce all manner of local and general disorders that vary with the idiosyncrasy (age, condition, mode of life, etc.) of the patient. By some the orbits are said to be shallow, with flattened and widely separated margins of the brachycephalic type; by others they are assumed to be both high and narrow as seen in the dolichocephalic skull.

The mould of the face is often characteristic. The bony features—particularly those around the broad, flat eyelids—are asymmetrical and not pronounced. The nasal bridge is depressed, the superciliary margins are flattened, and the facial borders recede quite irregularly, giving the subject a Mongoloid cast of countenance. With this peculiar physiognomy, there are a series of pathologic symptoms that are distinctive. The anterior chambers are shallow, the pupils are small, and the irides are active to the various forms of stimuli.

Habit-spasm, photophobia, hordeoli, and Meibomian cysts are all frequently seen. Dryness and marginal inflammation of the eyelids, that may become aggravated into squamous and pustular forms of blepharitis, which in the majority of instances are associated with microbic invasion, are also often present.

Thickening and irritation of the conjunctival membrane, with both stillicidium and epiphora, are not infrequent, while corneal disease of herpetic type, with and without episcleral disturbance, is not rarely seen.

In rapidly regressive cases particularly the chorioid, the retina, and the optic nerve-head bear their ophthalmoscopic testimonies. The optic disk at times exhibits a low grade of neuritis. The lymph channels of

the main retinal vessels are often thickened and opaque, while the venous stems may be somewhat engorged. The retina and the chorioid, especially in the region of the optic disk and the macula lutea, are not infrequently congested.

These symptoms, in association with the brilliancy of illumination, the great width of the ophthalmoscopic field, the low degree of magnifying power, and the necessity for an emmetropic observer to employ convex correcting lenses in order to render the direct image distinct, serve as a series of intra-ocular signs that are more or less expressive of the condition.

Certain forms of instrumentation applied properly and critically bring an additional number of objective signs into view.

Besides the direct method of ophthalmoscopy just alluded to, the indirect method, with its measurements of the relative sizes and situations of the inverted and reversed aerial images of the fundus-details, may be employed to advantage in the production of signs that are symptomatic of the error. A modification of the indirect method known as the fundus-image test is of comparative value in the evolution of several signs that are more or less useful. Here the movements of the convex lens give a series of symptomatic clues that may be of use in the determination of the grade of the error.

The fundus-reflex test, particularly with the plane mirror (using the concave one for verification of results), presents several most valuable signs that show the degree of refractive error to the greatest nicety. The direction and the rapidity of movement of both the shadow and the light, the intensity of the brightness and the form of the light area, and the positions of the points of reversal are the main signs that are brought into play by this method.

Dissection of a hypermetropic eye will show that the sclerotic coat is comparatively quite thick and tough, while the circular fibres of the ciliary muscle are generally hypertrophic.

**B. General Objective Signs.**—Besides the form of the cranium and the facial peculiarities, there are a series of general appearances brought into existence when such patients attempt to use their eyes for near work.

Work is commenced. A few vague expressions of discomfort, such as inward stroking of the eyeballs, appear. After a brief period of time the head is raised and the gaze is involuntarily directed for a few moments upon some distant object. The eyes are reapplied for lessening intervals of time and under increasingly greater illumination, only to be followed by a similar series of symptoms in increased force. Constantly varying the position of the object looked at, and not being able to obtain any distinctness of image, near work is laid aside, and either out-of-door life is substituted by the young and the sthenic; less disturbing employment sought by the unambitious and the pleasure-seeking; or, more often, particularly in the middle aged and the comfortably situated, irresistible drowsiness with unsolicited sleep soon appears and puts an end to all employment.

If, under some of the conditions, work be persisted in, as it frequently

is by such subjects as the mentally strong neurasthenic and the persistent skilled laborer, graver symptoms of reflex type exhibit themselves.

The more remote reflex phenomena that can be recognized objectively, and that are described at length in their appropriate places throughout the various sections of this article, are generally the results of combined conditions, such as astigmatism, imperfectly associated muscular actions, neurasthenia, toxæmia, etc.

C. **Local Subjective Signs.**—This subdivision includes many symptoms that are quite distinctive of the condition.

Uncorrected vision in young subjects, with low and medium grades of the uncomplicated variety of this form of refraction-error, is nearly always normal, and cannot be improved by artificial aid with convex spherical lenses. In fact, in many instances it is greater than that which is considered as the average. In the higher grades of the affection it is generally subnormal, while in the very highest—in which there is a probable imperfect development of the sentient portion of the ocular bulb—the retinal images become so small that the subjects bring objects closer to their eyes in order to make the images larger. At the same time they nip their eyelids so as to reduce the diffusion-circles,—these symptoms being quite expressive of myopia.

In the higher degrees of hypermetropia among the young, and in nearly every case of the refractive error in subjects who are beyond forty-five to fifty years of age (independent of senile sensory change), visual acuity for both near work and distance is reduced below the standard, and can be improved by the use of convex spherical lenses. Should distant vision fall below the average, the defect, when uncomplicated, is always associated with a recession of the near-point, this being especially pronounced during monocular vision.

Spasm of the ciliary muscle with its consequent refractive change is not infrequent. Ofttimes, dependent on the grade of the static error, it gives diverse forms of local subjective result.

Should the hypermetropia be of high grade, both near and distant vision may be improved. Should it be of low or medium grade, an inability to see clearly at a distance (transient or functional myopia) may arise. In some of these cases, a disturbing ocular neuralgia, which is made worse by efforts for convergence, is complained of.

By reason of the greater demand of the focussing apparatus of the eye during near work, the signs of lowered and imperfect near vision appear much earlier than those for distant sight. The symptoms most frequently present themselves in the form of recurrent momentary losses of near-focus dependent on sudden relaxations of over-strained action of the ciliary muscle with myodesopsia, which can be greatly relieved by the employment of convex spherical lenses.

The fields of vision and fixation are quite large, giving the individuals extensive areas for both direct and indirect vision.

*Types of Hypermetropia.*—The concealing power of the crystalline lens and the ciliary muscle on hypermetropia has given ophthalmic nomenclature three gross artificial varieties of the defect. The entire amount of the error is known under the term *total hypermetropia,* and has the symbol Ht to express it. That part of the total hypermetropia which is hidden by crystalline-lens action is known as *latent hypermetropia,* and is expressed by the abbreviation Hl. The portion that remains uncorrected and is exposed is designated as *manifest hypermetropia* (Hm).

In accordance with some authors, *absolute hypermetropia* (Ha) is said to be present when, in spite of the strongest convergence of the lines of vision, accommodation for both near *and far* vision is impossible. For the same authorities, so-called *relative hypermetropia* (Hr) and *facultative hypermetropia* (Hf) signify mere differences of ability of the two eyes to overcome undue efforts for convergence that are produced by overaction of the two ciliary muscles. The former expression is used by them when there is too great an inward deviation of one or of both of the eyeballs produced, particularly during near work. The latter term is employed to specify that there is an ability to continue a proper direction of the two visual axes during undue exercise of the ciliary muscles. In some of these cases, the subject can see as well with as without convex lenses.

Other observers employ different expressions for absolute and facultative hypermetropias. Starting with total and latent hypermetropia as stated above, they divide manifest hypermetropia into two parts. One of these, known as *absolute manifest hypermetropia* (Hma), is spontaneously exposed and cannot be naturally corrected by accommodative effort. This form requires the employment of correcting lenses to bring vision to normal. The other, known as *facultative manifest hypermetropia* (Hmf), is that portion which can be artificially evolved by correcting lenses, though their use is not necessary to retain normal vision. In many instances, particularly in young subjects, manifest hypermetropia and facultative manifest hypermetropia may coincide, while in older cases manifest hypermetropia and absolute manifest hypermetropia may be alike.

In all cases in which accommodation is lost, latent hypermetropia disappears, and manifest hypermetropia becomes merged into total hypermetropia.

*Neuralgia in Hypermetropia.*—Cephalalgia is most frequently distributed throughout the area of the primary branch of the trigeminus. At times it is referred to the apex of the orbits, and in neurasthenic subjects it may extend to the occiput. Generally beginning as a dull, heavy sensation in the frontal region, it, as a rule, gradually invades the entire nerve district and manifests itself in some localized area as a distinct pain. Variable, dependent in a measure on the character of the associated fault, appearing after prolonged periods of eye-abuse, often present on awakening, and worse during any exhausting process, such as disordered menstruation, mental anxiety, or undue fatigue, it becomes one of the most prominent symptoms

(in spite of the fact that it is unusual in the very young) that sends the sufferer to the medical adviser for relief.

D. **General Subjective Signs.**—Quickness of vision and, hence, rapidity of mentality, are at times seriously disturbed in young persons with low and middle grade hypermetropia.

## MYOPIA.

In myopia the symptomatic pictures are far different.

A. **Local Objective Signs.**—By some the orbits are said to be low, broad, and shallow in the young, increasing in height as the subject matures. The eyeballs are large, elongated antero-posteriorly, and at times are protuberant. The centre of motion is situated more deeply than it is in the hypermetropic globe and is quite close to the retina. Temporary exophoria soon manifests itself, followed later by various grades of permanent exotropia.

In axial myopia the angles *alpha* and *gamma* are narrow and in some pronounced cases they may be lost.

The summits of the corneæ are nearly always decentred in the middle and the high grade cases.

The conjunctival tissues are frequently injected, and there is congestion of the contiguous parts.

The anterior chambers, particularly those portions that are opposite to the pupillary areas, are deep. The pupils are dilated, and the irides are somewhat sluggish in reaction.

Ophthalmoscopically, the details of the eye-ground, which at times are seen in but limited areas, are greatly magnified and feebly illuminated. The optic nerve-head, the retina, and the chorioid evidence signs of low-grade inflammation, these changes being more noticeable in some of the temporarily progressive cases and in all of the permanently progressive varieties of the disorder. In these types a number of conditions in all grades and forms, such as temporo-papillary crescents, localized areas of disturbance, inflammation, and degeneration, dragging of the retinal vessels, and irregular stretching of the optic disk, with distortion of the porus opticus, constitute the main signs that are found in and around the optic nerve-head. Attenuation of the chorioid, ectasias and distentions of the scleral coat, and extension of the peripapillary atrophies, all add their appearances to the picture. At times fine macular changes, minute circum-macular swellings and thickenings, with almost imperceptible disturbances situated in the approximate portions of the hyaloid membrane and adjacent vitreous humor, can be noticed by skilled observers. In the graver cases, the vitreous humor becomes disorganized and is more or less filled with large freely mobile opacities, and the retina becomes detached.

In extreme cases, chorioidal and retinal hemorrhages with detachment of the chorioid take place. The crystalline lens becomes translucent, and at times is rendered opaque. Divergence is made permanent, and the organ becomes blind.

If the tissues of the eyeball be examined, the above changes will not only be proved, but it will be found that the circular fibres of the ciliary muscle are almost entirely, if not quite, wanting. It will also be seen that the ora serrata of the congenitally myopic eye, which has had but infrequent occasion for the accommodative act, possesses a fewer number of teeth (not the microscopic villous processes) than the constantly abused one of hypermetropic refraction.

During distant vision, the eyelids are nipped together in order to see more distinctly. The brow is wrinkled. The nostrils are updrawn and the head is projected forward. During near vision objects are held close to the eyes.

**B. General Objective Signs.**—Stooping is quite common, while hunching of the back is not rarely seen in old high-grade myopes of sedentary habits. Constantly seeking inefficiently lighted situations during near work and reading in a most peculiar way—by unconsciously moving the head from side to side—are quite characteristic signs of the condition.

**C. Local Subjective Signs.**—Normal acuity of vision for distance is never found in the uncorrected eye. As the expression "near-sight" signifies, normal sight is limited by a definite point which is used as a clinical measure for the degree of the condition.

Metamorphopsia, with enlargements of physiologic blind spots in the fields of vision, and ring-like scotomata, *muscæ volitantes,* and want of stereoscopic vision, are all more or less common.

**D. General Subjective Signs.**—Subjective signs of reflex disturbance from myopia are not pronounced. Low and moderate degrees of simple myopia are the least injurious of all the forms of ametropia upon the general health.

Unfortunately, however, asthenopia, that valuable (because ever-ready-to-cry-out) guardian, does not give its signals except in the high grades of the condition or in cases in which there is marked astigmatism.

Hour after hour is spent in abusing the eyes by taking advantage of greater ability to see close to the eyes; while reading or sewing by flickering firelight, during waning twilight, and with the aid of inefficient moonlight, is not an uncommon practice for this class of subjects.

The fields of vision are decreased in extent, this being particularly noticeable while the patient is wearing correcting lenses.

Depending on the gross configuration of objects, relying on the remembrance of style, color, and cut of dress, and intuitively taking advantage of previous knowledge of peculiarity of gait or of gesture of a distant individual, many myopes frequently surprise a bystander by their apparent ability to see correctly what has been recognized only by an unconscious feat of memory coupled with undue observation.

The mental evolution of a youth suffering from an unrecognized or a neglected myopia is instructive. Unable, by reason of deficient distant vision, to compete in out-of-door sports, and jeered at by his companions for his failures, he is early made to retire to the more pleasant tasks of

near work. Applying himself without any corrective for mental discipline and physical welfare, he lapses into desultory and miscellaneous reading, performed, as a rule, under the worst of circumstances. Unwisely applauded for his constant studies, he pursues his evil habits, which become increasingly fixed, until at last he becomes introspective and perverted in his tastes. The changing beauties of nature are unknown to him, and the varying facial changes and peculiarities of physical expression which represent the mental attitude of those whom he meets, are as nothing. As a result, self-consciousness, with the disturbing effects of repeatedly disappointing ideals, all too soon become a part of his mental attributes. Uncongenial to his fellows; superior to them in his knowledge, and thus arousing jealousy and its follower envy; timid to assert even known truths; and brusque and rude in order to hide a half-concealed fear of every new face: he finally leaves all manner of friendship, and, in spite of well-meaning opposition, seeks the worst thing that he can do,—constant use of the eyes to help fill his already over-burdened and badly trained mind, and sinks into sedentary life. Seldom practical, always in search of the metaphysical, and ever plodding but rarely applying himself, his mental vigor becomes so warped as to demand constant application. In fact, there is but very little true intellectuality in his make-up; his mentality is ofttimes a mere precocity, with physical deterioration set at a premium.

Such a subject, fitted only for a life's work in some mental groove that has but a few congenial spirits in it, and one that is ever demanding book-study, unfortunately will, unless the refraction-equivalent be rendered stationary by proper correction, hygiene, exercise, etc., suddenly find himself rendered unfit for a continuance of his work by reason of a catastrophe to the eyeballs themselves: the deplorable result of many such lives.

## ASTIGMATISM.

**A. Local Objective Signs.**—The total amount of astigmatism without any differentiation as to its exact situation can be evidenced objectively by instruments that are made to study the fundus of the eye. Most prominent among these plans are what are known as the fundus-reflex test and ophthalmoscopy in their different forms. In the former test the character, the grade, and the angle of the defect can be determined by the axes of movement and the angles of greatest and least reversal of light and shadow, and the variation of rapidity of the movement and the degree of reflex. In the latter the distinctness of various portions and positions of the fundus-details may be determined by the differences of amount of artificial lens-power that are necessary to be placed in various meridians of the sight-hole of the instrument, or the variation of distance at which the instrument must be held to see fine distinctive fundus-points that are situated at angles to one another. In addition to the former's optical determination, observation of a series of objective intra-ocular signs that are expressive of the refractive condition, are made by this second method.

At times the principal meridians of lenticular astigmatism will be found to be situated at right angles to those of any existent corneal astigmatism, this probably being dependent on a physiologic over-corrective influence of the crystalline lens on the faulty cornea. When the greatest and the least angles of the two forms of astigmatism coincide or when they are situated at relatively oblique positions, it is presumable that each is the result of an independent abnormality.

B. **General Objective Signs.**—The facial expression, with the head twisted to one side in accordance with the axes of the principal meridians of the astigmatism, the general attitude, and the mental characteristics of the subject; such as, for example, the peculiarities shown in the selection of dress-material, wall-paper, carpet, furniture-coverings, etc., evince not only the presence of this form of ametropia, but, to an observant clinician of experience, give a clue to the type of the special disorder. For example, the half-nipped eyelids of the astigmatic myope in his endeavors for distant vision are in contrast with the widely-opened palpebral fissures of the corresponding hypermetrope.

C. **Local Subjective Signs.**—Both distant and near vision in astigmatic eyes, no matter how slight the error may be, are rarely persistently normal. There is a latent astigmatism as there is a latent hypermetropia, which is hidden by the action of the ciliary muscle on the lenticular mass, and the disturbance when physiologically spoken of should be similarly divided into *total astigmatism* (As t), *manifest astigmatism* (As m), and *latent astigmatism* (As l). Although many cases of minor degrees of astigmatism have what is known as normal acuity of vision, yet the so-called test-card (and test-line) determination, in asserting that a vision of average normality 5/5 is perfect for any given astigmatic subject, may be false. Astigmatism, unless corrected by the ciliary muscle or by test-lenses, must reduce the vision of the individual; while the corrected perfection of vision may often rise to 5/2½.

The doubling of images so frequently seen may be in the form of a monocular diplopia or polyopia. Especially is this noticeable when an object gazed at is rather small and is not complex. As the vertical lines are the most important in the construction of letters, any form of defect that affects them must produce marked disturbance of vision. The fact, also, that to such a subject the hour-hand and the minute-hand on the graduated face of a clock or other timepiece assume different and distinctive visual values during their revolutions around the central post of the mechanism, partially explains why it is that complaints of inability to see well during certain periods of the day are not infrequently heard.

The plan of having the subject name quickly a number of distant letters of the size of type which represents his average visual acuity is an excellent method to make evident, by the characteristic mistakes obtained, the approximate angles of greatest and of least error.

Crystalline-lens action is so uncertain that the minimum amounts of

dynamic change which are able to maintain distant vision are so easily exposed that the least variation becomes manifest and alters the visual equivalent, offering to clinicians who are able to note the typical errors in test-type reading by such subjects valuable subjective data as to the nature of the condition. Radiary halos around localized light-areas extending to greater distances in certain directions are also often complained of.

Defective near-vision, which gives in the main only blurred, indistinct, and variable images of all close objects, is always disturbing and is most harmful if the possessor be at all neurotic. Here a whole train of reflexes that to the ignorant are irrelevant, and to the clinically experienced are quite apparent and interdependent, will soon appear: a succession of symptoms, which at first, though hard to recognize, are readily remedied, but later, though easily seen, are difficult to get rid of.

In spite of the assertion that depth of penetration practically corrects the faulty view obtained by an astigmatic eye when using the microscope, it is certain that, if proper tests be made, as, for example, by placing twelve perfect valves of a diatom in a spoke-like manner around a central area on a slide, any measurable amount of astigmatism can be recognized.

## PRESBYOPIA.

There are two types of symptoms in old sight: those that are connected with monocular vision, and those that are associated with binocular vision. To the first, which expresses itself by a recession of the near-point, and in many instances of the far-point also, is superadded—no matter how difficult it may be for clinical demonstration—a series of recurrent weaknesses of the exterior ocular muscles. These latter symptoms are particularly seen in monocular adduction and associated adduction (conjugate deviation) and, to a greater degree, during convergence adduction; a lessening of action that is not corrected by artificial focussing power added to the dioptric elements of the eyes.

If ordinary presbyopes are found to prefer to place their convex spherical lenses further away from their eyes by allowing the bridge or the spring of the frames to rest nearer the tip of the nose, it is certain that the lenses in use are two weak for their intended purposes.

The pupillary contraction of age, as can be well understood, serves as an advantage to the presbyopic subject, and hence he is frequently found facing the light during attempted near work.

So-called "second-sight" is generally complicated with astigmatism which is contrary to the rule. Cases of this kind frequently exhibit thickenings and inequalities in the density of the substance of the lens with slight peripheral lenticular opacities.

## APHAKIA.

In some cases of aphakia the subject, contrary to all beliefs, apparently possesses the ability of focussing. This is probably accomplished in a meas-

ure either by an impulse of convergence adduction producing pupillary contraction or by the continuance of the habit of pupillary contraction acting on the ciliary muscle, and thus decreasing the size of some stenopæic openings situated in the opaque capsule remnants that are left in the pupillary area, the result being a disappearance of diffusion circles, with the ability to focus at any point along the visual axes at which the object is able to form a retinal image of sufficient size to be distinguished. This partial explanation can be proved clinically by observing the ability of normal subjects, who, while having full ciliary paralysis and a dilated pupil from any reliable cyclopegic and mydriatic, are able to focus for both near and distant objects through pin-hole openings made in opaque discs that are placed before the eye.

The explanation given by some that such focussing is accomplished by an "individual ability to interpret dispersion circles" is not at all certain. The very presence of minute slits and round openings in the opaque capsule in such cases is not only confirmatory of the former supposition, but is ofttimes sufficient answer in itself for the power.

In aphakia that part of visual indistinctness which is dependent on corneal haze and irregularity may be made objectively manifest by careful examination with oblique illumination.

## HETEROPHORIA.

Ordinary muscle-equilibrium is found to be one or even two degrees of latent heterophoria (exophoria in myopes and esophoria in hypermetropes) for about five or six metres' distance, with very slight exophoria at thirty-five centimetres' distance on the median line. This is seen in cases in which the astigmatic axes are even situated at ninety degrees; the rule being that when the astigmatic axes are obliquely placed or when the relative amounts of the defect are unlike, the muscle-error is the greater and the more exposed. The deviation in the vertical direction, though much less marked, is much more troublesome.

Heterophorias soon become apparent (Het m) with the minor degrees of ametropia, particularly of the astigmatic variety. Latent exophorias are often made manifest by the use of prisms and the repeated performance of so-called graduated tenotomies. Hyperesophorias are quite common in young highly hypermetropic and astigmatic subjects, while hypoexophorias with slightly dilated pupils are the rule in the middle and the high grade myopes. The extra-ocular muscle imbalances are the greatest in antimetropic and anisometropic subjects.

In addition, temporary and changeable astigmatism, which is lenticular in character, may not infrequently serve as the interior expression of an inequality of exterior muscle equilibrium.

The terms ordinarily used to express muscle-balance will, in a measure, be employed. *Orthophoria*, or a proper tending, in which there is no tendency to deviation in the primary position, means a normal muscle-balance. *Hete-*

*rophoria*, or irregular tending, signifies, in opposition to heterotropia, an improper muscle-balance that can be constantly overcome by muscular effort.

In accordance with the principal directions of the main deviation, heterophoria has several general subdivisions,—*esophoria*, in which the main tendency is directly inwards; *exophoria*, in which the main tendency is directly outwards; *hyperphoria*, in which the main tendency is upwards, and *hypophoria*, in which the main tendency is downwards. The term *antiphoria*, in which the directions of the main tendencies of the two eyeballs are opposed to one another, may be employed. Like ametropia, heterophoria is divisible into *manifest* (Het m), *latent* (Het l), and *total* (Het t).

As all of these special forms of deviation are the results of general extra-ocular muscle imbalance, it becomes in consequence difficult to draw any scientific line between them. For example, at what mathematical point or in what geometrical position the divisional line between heterophoria and heterotropia can be made is, like many other ophthalmic empiricisms, undefined as yet, though, for all practical purposes, it can be stated roughly that, if diplopia should appear spontaneously at times, or even during the placing of an ordinary red glass before one eye, while both eyes are made to gaze at a distant point of light, the case may be considered to belong to the second category, the disturbance most probably having passed through the heterophoric stage. This, it must be understood, is independent of a spontaneous doubling of images that is dependent on differential innervation, which can be experimentally determined as normally occurring upon marked excentric binocular fixation, particularly in the vertical field.

*Local Effects of Ametropia and Heterophoria.*—The local signs of the inward tendencies are, as a rule, those that were shown in the description of etiology, etc. These are particularly noticeable when there is an associated astigmatism: so too with the reflex groupings. The local symptoms of external deviations are frequently (as studied in detail in the beginning of this article) those that have been shown to be dependent on underaction and paresis.

An odd symptom, and one which is well established as one of the rarer signs of heterophoria, is the sensation of a sudden impact against the eyeball, produced probably by a simultaneous action of two or more of the rectus muscles.

Undoubtedly, reflex ptosis may be caused by hyperphoric disturbances. In some cases of hyperphoria a tilting of the head away from the shoulder corresponding with the hyperphoric eye is said by some to take place, the result probably of torsional influences. Palpebral hyperæmia with watering of the eye, diminished vision, and confusing double vision, as seen in "car-sickness," etc., with all manner of general reflexes that are more or less distantly related, constitute some of the other main signs of hyperphoria.

So-called "cyclophoria," or rotary tendency, is at times found as a clinical expression of imbalance of equilibrium and action of the exterior ocular muscles. It is most frequently seen in "oblique astigmatism," and is con-

sidered to be peculiarly related to disturbances that are mainly connected with the oblique muscles.

Facial characteristics, consisting of constrained actions of the muscle-groupings which are situated around the eye, the nose, and the mouth, resulting in wrinklings, corrugations, and even grimaces, that are in a great measure symptomatic of the types of special heterophoric disorder, become at times quite noticeable.

*Effects of Ametropia and Heterophoria on the General Health.* — A variety of general symptom which is not noticed until a careful correction for ametropia or heterophoria is properly placed in position, is an unexpected sense of well-being, due to a getting rid, as it were, of an undue expenditure of nerve-energy, which, if it had not been thus removed, would have continued its injurious influence upon the general health.

On the other hand, it must not be forgotten that general organic lesions, particularly those that are found in neural structures, may give rise to similar signs which are situated in the motor portions of the visual apparatus, and which to the untrained physician are quite confusing.

The form of ametropia that is the least injurious to the general health is a moderate grade of simple myopia, but this condition is rare. The same is true to a less degree of compound myopic astigmatism. So also with low degrees of hypermetropia, especially as found in the young. The moment, however, that ametropia becomes pronounced and anisometropia and antimetropia with astigmatism (no matter of how slight a degree) are brought into play, mischief appears. Especially is this so when the astigmatism is of mixed type or when the principal axes are situated against the rule or are oblique and asymmetrical: then the reflex disturbances are usually at their utmost.

## PART IV.

### PROGNOSIS.

With proper care and adequate rest, much good work can be secured from the visual organs. Healthful and recuperative exercise signify sound tissue. The moment that friction begins, mischief manifests itself, which, if it be unrecognized or uncorrected, is apt to produce physical disorder.

Although ametropia cannot be cured, its effects are apparently so slight that they have no major place among the statistics of blindness. Hidden and ofttimes unwatched, it slowly expends its harmful impulses on the delicate visual matter without being cared for: and too frequently, when recognized, it is so imperfectly corrected that its detrimental influences are increased and its evil consequences become more widely spread.

In many asthenopes who lead sedentary and enervating lives, the weakened sight is but one of the evidences of general nerve-tire. The exercise of the normal sensory act during every waking moment would ofttimes proceed uninterruptedly and unharmfully when combined functioning for

near objects takes place, were not the general weakening factors constantly at work. In such cases the merest departure of the ocular structures from a normal condition may produce a whole train of faulty and injurious responses.

*Effects of Asthenopia.*—Asthenopia never received the attention that was due to it until about two decades ago, and then only in a country in which the ravages of near-sight had become so apparent as to render an immediate reform necessary for the well-being of the nation. There the injurious effects had become so disastrous to the community that arbitrary hygienic laws became imperative. Here, in the United States of America, having had this object-lesson given by an older nation, the evil, through similar enactments, constant supervision, and protection of eyes ailing to the slightest degree, has been to a great extent avoided. Statistics, observations by competent authorities, and the records of optical establishments, show the results of the work of reform among the native-born Americans: work that is being carried on to the same degree of usefulnesss as vaccination and asepsis.

Though this be so,—notwithstanding the fact that the prevalence of myopia, for example, has been lessened,—the other disturbance of the greatest prognostic importance—the want of harmonious binocular action—has, until the past decade and a half, remained generally untreated. Here, in a nation in which nerve-energy is so heedlessly wasted and even climatic agencies seem to be at work, has been found asthenopia at its worst. The rush for riches, the fight for fame, the sacrifices for social position, and the want of exercise and repose, have been the main general factors in the production of the condition.

Such a complex organ so situated must, as its work has continued, have evidenced further local changes and have become the cause of harmful influences on distant, though at times not well-understood, related organs.

*Effects of School-Life on Asthenopia.*—A glance at many of the scholars of the private schools of the past affords an excellent illustration of the causes and the effects of asthenopia. Taken from the better classes, more delicately nurtured, more dependently trained, and less able to withstand physical disturbances, they lived, as a rule, in old-time mansions that were devoid of comfort and proper sanitation. Crowded in badly ventilated rooms and allowed unhygienic and contaminating freedom, they soon began to show the signs of general weakness with eye-strain.

After being rushed through such a school-life, the real life-work was begun. By reason of physical unfitness the necessary duties were accomplished only under repeated strain until, as the most important part of the general disturbance, the ocular apparatus became inadequate for the performance of its ordinary functions.

With the brain-laborers the results were the worst. Obtaining their livelihoods by the exercise of their most delicately contrived sense-organs, hurried in the ingestion of their food, living in overheated and vitiated

atmospheres, and neglectful of health-giving exercise, they pursued their vocations under the most adverse circumstances. In them neurasthenia soonest became manifest and the eyes were among the earliest organs that became involved.

As with the higher schools, so to a greater degree with the so-called kindergartens. In these, unless properly guarded against, asthenopia began so early that radical changes in the ocular structures were liable to take place. To regulate play, so as properly and logically to exercise the developing and the growing sense of the infant-mind with the least harm to the organs at work, should have been the first principle in the "gardens of children," as Friedrich Froebel so happily designated them. For all kindergarteners to have obeyed Froebel's object,—"to give the children employment in agreement with their whole nature, to strengthen their bodies, to exercise their senses, etc.,"—much was required. To have done this without harm to the eyes, there should have been frequent intermittences in work. All exercises that were necessarily made close to the eyes should have been pursued in the diffuse sunlight and if possible while the children were in the open air.

Play-studies should have been given when both the physical and the mental powers were at their best. Search should have been made into hereditary taints, evil influences, and harmful tendencies, and all these should have been duly corrected if possible. Injurious ocular habits should have been persistently broken by the substitution of counteracting influences.

Fortunately, at present, here as elsewhere, the most harmful of these evils have been removed, as the results of careful study and conscientious correction from competent medical men.

By thus placing healthy subjects in good environment, etc., and giving them work which increases the normal power of their visual organs, together with exercises that bring as few as possible of the movements of the muscular apparatus of the two eyes into action, every individual may have given to him the best physiologic action with consequent improved mental mould, without interference with his ocular structures.

In all school-work the visual apparatus needs examination by those who are competent to give intelligent opinion and judicious advice. Those schools in which eyesight is considered during entrance-examination and for each successive term should be given the preference.

*General Effects of Asthenopia.*—The effects of impaired and difficult vision on the intellectual development and the physical growth of the subject are marvellous. Ofttimes unrecognized until the symptoms betoken alteration in the eyeball, warning comes too late, and the sufferer finds it necessary to be a slave to a pair of correcting lenses. Being by many physicians regarded as unimportant, the ocular disturbance frequently remains untouched, allowing the condition to become more pronounced, and hence increasingly harmful.

From a utilitarian stand-point, the broader the mental horizon of the individual is made the more useful does he become to others. As illustrative, take the case of his Imperial Majesty Nicholas I. of Russia, who was an imperfectly corrected myope with a far-point of but thirty-five centimetres. His ametropia helped give him a mode of life that had an effect on the geographical face of Europe lasting to this day. Physically superb, reared in retirement away from companionship, introspective to a fault, not permitted to engage in open combat, though both brave and obstinate, narrowly pious, fanatically despotic, remorseful, and disturbed to the hour of his death,—here, in spite of a fair heredity, were all of the mental and the moral characteristics of a faultily corrected brachymetrope—a "myope" holding in control the lives of millions.

*Emmetropia and Asthenopia.*—Although it is a theoretical doctrine that a pair of young emmetropic eyeballs do their combined work the easiest and with the fewest injurious influences, yet their great rarity makes them a something to be sought for rather than that which can be obtained. Practically, there cannot be any ocular ideal; and the ideal or emmetropic condition sought for may not be that which is fitted for some particular pair of organs. Moreover, an eye cannot be expected to remain permanently emmetropic when its dioptric media are so highly changeable and when its muscular movements are so indeterminate.

*Significance of Therapy on Ametropia.*—If it be true, as a French statesman has calculated, that an eye travels about a mile in reading an ordinary book, great care should be taken, in order to prevent any serious consequences, to see that everything used during any form of near work should be the best that can be gotten; as such preventive medicine, as it were, will do more to keep the eyes free from gross disease than any form of therapy. Asthenopia, however, is frequently a blessing in disguise, not only for the possessor, but often, if early seen and removed, for the offspring.

Lens-therapy, just as with any other form of therapeutics, should be subject to legal regulation. The evil of allowing certain ocular conditions to remain unrecognized is most reprehensible, as the symptoms disregarded may be expressive of local conditions which, if left untreated, will probably result in irretrievable blindness, or may be peculiar to dyscrasias that might be stopped in their incipiency, before the ravages of the general disease become so great as to lead to irremediable results.

In fact, it would be far less dangerous, and by no means so unethical, for a man to attempt to guide a valuable and a richly freighted vessel across the seas without the latest information in regard to the laws of storms, tides, and currents and a thorough knowledge of everything that pertains to his craft, than it is to take into his hands a series of apparently innocent tools with which, for mere monetary gain, he is allowed—and by some even solicited—to attempt to grant certain inadequately understood wishes, such as increase of visual acuteness, greater ability of near-focussing, etc., while the true mischief-maker remains unsought for and untouched.

The view from a non-professional stand-point that a glassed employé exhibits a confession of weak sight is wrong. The truth is, that he who is properly corrected, as a rule, enjoys better and more comfortable vision and, in consequence, performs a higher grade of work with greater ease and more rapidity than his uncorrected fellow-workman.

*The Effects of Hygiene on Ametropia and Asthenopia.*—The return to out-of-door sports, rendering school-life in the United States of America and Great Britain more healthful to the student, has, by lessening the hours and the amount of study, decreased the degree of gross ametropic change and lessened the gravity of the prognosis of the condition. This has become so well understood that in some of the more important institutions steps have been taken so to grade and to regulate the preliminary training for a broad education that it may be adapted to the work of the chosen after-pursuit and intended life-work.

The oft-repeated demand for specially adapted schools for the ametropic would be useless were the question of ordinary school-hygiene, personal supervision, and examination of the ocular condition of all the candidates and scholars duly considered by competent authorities. School-directors should be persons of intelligence and education. They should be well informed as to the construction and the maintenance of school-buildings. Women, who are brought much more closely into contact with children's wants, should be placed on such boards. Teachers should be required to understand the subject of ocular hygiene. Tracts descriptive of the care of the eyes should be distributed among parents and guardians and made a part of the studies. Object-lessons and illustrated lectures should be more frequently made use of, while the hours of study should be radically lessened: these done, the prognosis of ametropia would be greatly bettered.

Fortunately, much of this has come to pass, particularly in the great public schools of this country; though, in view of the increasing amount of prolonged eye-work that is being imposed on developing and growing children, it would be better in many departments of study to return to the oral teaching of the ancient Greeks.

Municipal statutes should be enacted in regard to proper examination of the ocular conditions of every candidate seeking entrance and of every scholar seeking advance, both in private and in public schools. Were this done, the children would be more suitably equipped and much better protected and, as a consequence, there would be a greater probability of their becoming good and useful citizens.

It is certain that the careful correction of ametropia, particularly that of astigmatism, has lessened the proportion of cases of high-grade myopia. The number of such cases at present applying for treatment in this part of the world is much less than it was only two decades ago. Disastrous intra-ocular changes associated with high myopia are not so frequently seen at the present time. The orthopædic holding of the morbid state in abeyance began some years ago to manifest its good work on the masses. The

process, slow in its progress, shows that here in the eastern portion of the United States of America we are on the eve of a radical change in the relative proportion of the different types of ametropia. The prevailing form in those cities in which careful work is done is that of a low grade of compound hypermetropic astigmatism. The national type is probably a somewhat greater degree of the same form of error. Germany, France, and England, with their now better practical knowledge of the etiology and the prevention of myopia than formerly, have also reduced the prevalence of the ametropic condition of gross harmful types to much lower percentages than previously. Their good work has not only saved the eyesight of their own people, but indirectly has greatly bettered that of the world at large.

Comparison of the relative frequency of disturbing ametropia of the past with the present, by reading old records of well-described though imperfectly appreciated clinical accounts of obscure cases, makes it evident that many more children of the previous generations would have worn correcting lenses had there been a proper knowledge of the subject. More recent statistics evince a fact which is well known to any working ophthalmologist of large experience,—that during the past decade in this country more children wore glasses than at present. This finding is dependent not only on certain well-known facts, but also on the fact that the children of that time inherited ocular tendencies which had been uncorrected and had even become malignant in not a few instances among their progenitors.

*Significance of the Myopic Eye.*—That a myopic eye is a direct accompaniment of advanced civilization, there can be no doubt; but for many reasons, this change of ocular condition cannot be attributed to any such embryologic cause as that, the sentient portion of the eye and the brain being originally derived from the same derm, the increased growth of the one means the relative increased growth of the other. The eyes of those who have been mentally the most active in special pursuits requiring accurate and prolonged near vision from early life, and who have done the greatest amount of intellectual good, have not necessarily been myopic in character.

In opposition to this (*i.e.*, the low and the medium grades of uncomplicated myopia), the grossly myopic eye is, almost without exception, a diseased organ; one that has been recruited from the hypermetropic lists, from eyes that are more or less inadequate for near work. It is an eye which, in the very nature of things, is almost invariably possessed by subjects who, never having taken much exercise and having lived sedentary lives, have become physically weak and unable to withstand much strain on their working tissues. The prognosis in such cases is not only dependent on the care of the ocular structures, but is in direct proportion to general hygiene and therapy.

*Statistics in Regard to Ametropia and Asthenopia.*—Study of hospital and dispensary reports shows that the presence of astigmatism—the most important factor in the prognosis of ametropia—is less frequently noted than it is in private practice. That this is merely apparent can be under-

stood when it is considered that, as a rule, such public work throughout the world is done with too great haste and, hence, with inaccuracy; and, further, that it is relegated to beginners, and hence to those who are the least experienced.

Simple uncomplicated myopia is so rare that its real percentage of existence is reduced to a fractional amount. Care in the determination of astigmatism will lessen the statistics as to its prevalence that are so constantly offered. In this connection, however, it must be remembered that if clinical statistics be taken as a criterion as to the actual amount of myopia in existence, many cases of the lower grades of the error will be omitted, as they fail to present any or but little asthenopia.

The statistics of two per cent. of complaining cases of ametropia that are free from astigmatism is uncertain. Experience has shown that the only statements in regard to the presence of astigmatism that may be considered accurate are those which have been obtained during total cyclopegia.

If disturbing astigmatism be so prevalent and constitutes such an important factor in the lessening of hypermetropia, the increase of myopia, and the production of heterophoria and heterotropia, it is evident that its early and full correction must help arrest the stretching processes of the eyeball, and in time cause nearly all myopia, except that which is either congenital or one of the results of gross ocular inflammation, to cease. Considered in this manner, the correction of astigmatic faults serves as an agent of the greatest prognostic value.

Again, as it is certain that the minor degrees of ametropia and heterophoria produce impairment of nerve-energy by an abnormal expenditure of nerve-force, the earlier such cases are corrected, the fewer the chances will be that the nervous impairment will be made worse, become permanent, or allowed to give rise to organic disturbance.

Were the human race cyclopic, the problem would be easier of solution, and eye-strain would be less disastrous in its consequences.

## PART V.

## TREATMENT.

### GENERAL CONSIDERATIONS.

It can be now understood that, practically, a normal eye is one that has healthy tissues and is functioning properly. It need not be emmetropic: neither need it be exactly like its fellow. If proper physiologic action can take place without strain or injury, and if the physical material of the organ remains undisturbed, normality of both action and tissue can be assumed. The moment that wear is conver'ed into tear, use into abuse, and the abused tissues express their disturbance by faulty and painful physiologic response, eye-strain has announced itself, and the therapy of rest by

disuse, orthopedy by lenses and prisms, and betterment of general condition, must be applied. Visual improvement becomes but a part of the treatment, while the ocular disease is the main condition that must be removed.

Imperfect sight and uncomfortable vision without any apparent inflammatory signs or acute ocular pain are the two principal symptoms that send the ametrope for relief. The concealment of the principal objective signs of the disorder by the overlying coats of the eye, and the almost universal belief that all defective vision except that which is associated with acute ocular disease can be relieved by lenses, act disastrously on the welfare of the organ. The structures are so small and the inflammatory reactions provoked by ametropia are so slight that they often fail to present any gross symptoms which can be detected by an uninformed physician or layman.

The long-continued professional habit of merely attempting to relieve defective vision without inquiring into its cause, the ordering of glasses by those who are engaged in the manufacture or sale of optical instruments, and the want of a broader dissemination of knowledge of the true bearing of refractive changes on the health of the visual apparatus and the well-being of the individual, are a few of the reasons why the estimation and the correction of ametropic fault have so often been left by the public to uncertain empiricism.

The increased knowledge of both the general medical profession and the laity to degrees of information that are consistent with a proper combined understanding of the subject, and the rapidity with which the most useful forms of instrumentation have appeared and the most accurate methods have been applied, have aided in setting blundering ignorance, trade prejudice, and mercenary desire to one side, and have helped both the physician and the mechanician in their true relative positions,—the one who, seeing and understanding with a medical knowledge, prescribes what should be used; the other who, skilled in the making and the fashioning of delicate instruments, contrives and constructs the desired appliances and adjustments.

The science of prescribing for ametropia is not, as has been so widely supposed, a mere mechanical art, similar to the fitting of gloves and the measuring for boots. It is the work of the educated physician, who, understanding the significance of eye-strain, its bearing on the general health, and its proper medical treatment, is able to care for the case. It is he who should be the one to decide for the patient. It is he who is the one upon whom responsibility for the individual not only rests, but who exerts also a far-reaching influence upon men at large and their actions.

In its treatment the eye should not be considered as a mere mathematical or geometric contrivance governed by optical laws that necessitate mechanical adaptation of focussing materials similar to those used in the various instruments of optical precision. The visual apparatus must be thought of as one of the physical structures of the entire organism, and subject to the same laws of physiologic action and disease as any other organ in the

economy. The visual bulb is an end-organ, and not simply an optical contrivance, like a microscope or a telescope, to look through. It is a living mechanism wherein rays of light are changed into a physical material that is capable of being evolved into perceivable matter. The popular comparison of the eye to a camera is erroneous and harmful.

The difficult or impaired vision that the patient comes complaining of is but one of many symptoms—the most prominent subjective one—that constitute the signs of the disorder, so that in the treatment of the condition optical correction becomes but a part of the therapy, and should be ordered only by the competent medical man: not competent merely in the sense of being one who possesses a medical diploma, but competent in the fact of having had a broad medical education and sufficient knowledge of diseases of the visual apparatus to treat them understandingly.

The employment of a correcting lens in ametropia is just as much a part of medical therapy as the use of a crutch for talipes, a brace for scoliosis, a splint for coxalgia, and a truss for hernia, and is to be prescribed as any other appliance or drug by those who are legally fit and hence lawfully responsible for the same. The lens, the crutch, the brace, the splint, and the truss, each just as any drug, should be made or prepared in accordance with definitely formulated rules, arranged by a board of medical governors and given to the profession and trade in some authoritative way like the "Pharmacopœia." Under such rules no lens, any more than any drug, could be ordered by any one not understanding its action and its indications for employment; the work of the medical man alone, and one that no conscientious and well-educated mind would think of assuming until a course of legitimate medical study, a foundation of broad practical general medical work, and a sufficiently prolonged and constantly repeated exercise of special training, had all been thoroughly and painstakingly given to it.

As with every other therapeutic aid which has much good in it, its virtues were at first exaggerated, and correcting lenses were frequently ordered in cases in which there was no real necessity for their use. This excess, fortunately, has now become almost a thing of the past, thus placing the method upon a proper and a specially useful basis.

Properly educated, no layman having any respect for his physical condition would for a moment imagine himself able to prescribe for his eyes should he find an impairment of his visual functions. Realizing that the defective sight was but a symptom of a physical change that must be medically determined and understood before it is to be corrected, he would not select his own remedy for the visual defect. Treatment would then be sought by him not merely from a desire for betterment of sight and the temporary relief of asthenopia, but also in order that he might have his visual apparatus placed in as normal a working condition as possible, so as to preserve the physical structures to their uttermost and indirectly relieve all related disturbances. He would understand that if therapy is to be successful, it must, as a rule, be dual in character: first, to remove any

local disturbance, and, second, to rid the general economy of any harmful relevant conditions.

The question is not one of a mere mechanical adaptation for optical assistance, but it is the higher medical one of conservation of vision with protection to the visual apparatus and betterment of the general health.

*Special Study of Ametropia.*—Cases coming for the correction of ametropia as a therapeutic measure constitute at least eighty per cent. or more of the patients that are seen in practical ophthalmic work. Of this percentage, among the young and the middle aged, at least sixty or seventy-five per cent. is found in the female, this probably being due in a measure to a greater demand on the eyes of such subjects for applied work, with an increased sensitiveness of their nervous systems and inferior powers of resistance.

The asthenope of to-day, unlike his ancestors of but one or two generations ago, is not authoritatively doomed to pursue a life of comparative uselessness in an out-of-door existence. No more is he condemned to "pastoral pursuits" or more brutally told "to go dig cellars." Now, on the contrary, scientific medicine has advanced so far that, should such advice be given, it would in the vast majority of cases mean professional carelessness or ignorance—the one ethically wrong, the other criminally negligent.

Cure in any of the forms of static ametropia, except by surgical procedure in a comparatively few instances, cannot be expected. There is no specific. The conditions can only be corrected. Therapy is preventive and orthopædic in type. On the contrary, the dynamic and the functional varieties of the condition are susceptible of cure both by local means and by general methods.

In the majority of cases the eyeball is badly shaped, in which event the correcting lens simply serves a purpose similar to that of the helping crutch in the case of a shortened or crooked leg. This constitutes the orthopædic part of therapy.

Hygiene and preventive medicine have also done much to keep the morbid processes in abeyance, and have tended to restore the visual organs to useful working capabilities without any influence on their tissues.

In all lens-therapy, however, it must be remembered that, just as there are changes in the form of the hygienic measures which are employed, as there are differences of medicinal dosage, and as there are peculiarities of construction in other orthopædic apparatuses, so it must be understood, both by the patient and by his medical adviser, that any given pair of glasses cannot be expected to remain permanently efficient, and should be changed whenever necessity requires. The amount of the correction of refractive error of to-day may not be that of the morrow. Like the drug, it should be given therapeutically, as it were, and ordered in strict accordance with circumstances.

As a rule, lenses and prisms should not be used in order to better vision alone, but should be employed either to prevent the progress of the disturb-

ance or to remedy a disordered mechanism. They are to be considered as curative agents *per se*. Literally, it is a visual apparatus that is being dealt with, and not an eye-strain, which is a symptom, or an ametropia, which is a special condition. How true this is can be better understood when it is considered that not infrequently it may be necessary at first to lessen vision in order to remove a harmful symptom-complex from which a subject may be suffering.

Glasses should seldom, if ever, be ordered to be worn during the active stages of ocular inflammation. Correcting lenses should be worn the moment that competent and conscientious advice prescribes their use. It is not the amount of the ametropia, particularly astigmatism, which determines the necessity for artificial lens-correction. A cylindrical lens of an eighth of a diopter strength has frequently done more good as a therapeutic measure in changing a muscle-imbalance to normal equilibrium in a pair of ametropic eyes, and thus ridding their possessor of a troublesome asthenopia, than has been accomplished by any general treatment or by so-termed rest-lenses (weak spherical lenses). All refractive error, especially the minor degrees of astigmatism, should be carefully estimated for at least as low a strength as the one-eighth of a diopter power difference, before it can be understood that the ametropia has been scientifically and fully estimated. (In this connection it is of interest to know that convex lenses of a half-diopter strength were of recognized value and in public use during the early years of the present century.)

Unfortunately, the very subject who has the minor degree of ametropia is, as a rule, the one who complains the most that he cannot take the time for proper treatment. He sees so well that he feels that he cannot be spared from work. He says that he must have his orthopædic help obtained as quickly as possible. A few emphatic denials of compromise in any uncertain way will soon convince him of the foolishness of the request. Understandable illustration and apt comparison with other therapeutic methods will show him the best and the most certain of the various procedures.

It is the total error, particularly the astigmatic one, that must be ascertained before a proper relationship between it and the amount that should be employed can be established in the giving of orthopædic help to the combined two organs.

Although over-nice distinctions between good and bad are, as a rule, useless, yet in the correction of ametropia "almost right" is of a more serious nature for the patient's good than "greatly wrong." There can be no other rule than absolute certainty in all ametropic estimation, in spite of the daily practice of busy medical men, who, through excuses of waste of time, etc., endeavor to dissuade their patients from measures that are necessary to obtain the most certain of results. In all subjects that are younger than forty-five years of age (and even in some instances up to fifty years) there must be as full paralysis of the ciliary muscles as possible. All else is but approximation. In regard to this, however, it must be remembered

that slight amounts of uncorrected lenticular astigmatism caused by irregular action of the radiary fibres of the ciliary muscle are said to be left after the use of an ordinarily efficient cyclopegic, the drug, it being asserted, having no control over this portion of the muscle.

*Cyclopegia and Cyclopegics.*—The rule governing the choice of the cyclopegic is to select one that will produce total cyclopegia for a length of time that is sufficient for the employment of those objective or subjective plans that may be deemed best adapted to the particular case. If the drug is to be used simply to estimate the ametropia, it should be the one which gives full paralysis of the ciliary muscle without any local or general complication, and the one the paralyzant effects of which pass away in the briefest period of time possible. If an additional therapeutic action be desired, the drug giving the best results with the least amount of local disturbance and constitutional effect should be chosen.

At times certain cyclopegics act irregularly on the ciliary muscle, and even during the early stages of their action may provoke a spasm, which is relieved either by the employment of more of the same material or by the substitution of other drugs of the same therapeutic type. In these cases the pupillary area will often temporarily assume the most peculiar shapes and changes of angulation. Total cyclopegia, however, cannot be assumed from apparent full pupillary dilatation. Tests as to the existence of any remnant of accommodative power left must be made before complete ciliary-muscle palsy can be asserted. At times, in certain cases of ametropia with reflex symptoms, such as neuralgia, chorea, gastric disturbances, etc., an instillation or two of some sufficiently strong cyclopegic will often, by a persistence, a lessening, or a disappearance of the general symptoms, be of use in the determination of the question of interrelationship.

To obtain the best effects of solutions of cyclopegics, they should be so dropped on the upper corneal margin as to flow over the surface of the cornea. Care should be taken to have the lower conjunctival cul-de-sac fairly dry, and to see that the lacrymal puncta are compressed or everted.

If cocaine is added to any cyclopegic in order to strengthen its action, the eyelids are to be kept closed for some time after the employment of the drugs, this being done in order to prevent any disturbance of the epithelial layers of the cornea, which would tend to defeat the purposes for which the drugs were employed.

During the use of the cyclopegic, by reason of its mydriatic effect, smoke-tinted glasses should be used in order to lessen the glare of incoming light. Whether they are flat or are made *coquille* in shape, it must be certain that they are free from blemish and that they are devoid of lenticular or prismatic action. If dark glasses cannot be obtained, two or three folds of a fairly narrow-meshed veil can be placed before the eyes, or, as a last resort, the experiment of blackening the face on and around the eyelids may be tried.

In some cases in which there is irregular astigmatism, the mydriatic

effect of the drug renders the results of subjective measurement uncertain. In such instances it is best to attempt to obtain a series of similar answers while the pupil is gauged to its ordinary size during use.

It may frequently happen, in irregular astigmatism or in some cases of regular (particularly oblique) astigmatism, that the objective findings will vary from the subjective results, this dissimilarity being probably dependent on the habit of the individual to employ an eccentric and more symmetrical part of the corneal membrane for ordinary visual purposes.

So with the various methods for the objective determination of the exact positions of the meridians of greatest and least curvatures of the cornea and the lens. These plans could be always depended upon in the correction of ametropia if it were known what the subjective choice or the selection of angle might be during binocular vision.

*Miotics.*—The employment of so-called miotics to lessen more quickly any cyclopegic and mydriatic effects is practically useless, as the action of such drugs is too weak, too spasmodic, and too evanescent, when used in amounts that are comfortable to the eye.

*Correction of Related Heterophoria.*—To relieve asthenopia it is just as important to obtain definite data of heterophoric changes, by repeated and properly gauged objective and subjective tests, as it is to determine ametropia to its minutest degree.

The impossibility of artificially controlling the extra ocular muscles, as is done to the intra-ocular groupings by some trustworthy cyclopegic, renders it impossible, in the present state of medical knowledge, to determine accurately the total amount of heterophoric action (particularly the latent heterophoria) in any definite position in the same way as latent ametropia is brought to light; though clinically, the muscle-equilibrium for distance may be often advantageously tried while the ciliary muscle is artificially paralyzed.

Prismatic corrections, like spherical and cylindrical ones, have their limitations. During their use, if it were possible, just as for the correction of ametropia, there should be definite modifications of power for every finite point used. Unfortunately, in such corrections there are the most troublesome factors of identical points that are situated throughout the associated fields of vision.

No therapy of any kind should be applied to the extra-ocular muscles until the ametropia of the two eyes has been scientifically and fully determined and relatively corrected. In fact, before any form of therapy can be employed, the powers of the combined muscle-actions in at least four of the principal directions (adduction, abduction, hyperduction, and infraduction) must be learned in order to understand the relative strengths of the different movement series. It must not be forgotten that the combined actions of the two ciliary muscles also enter into the question, thus necessitating a careful study of their interrelationships.

In brief, it is a series of motor impulses in an unstable muscular ap-

paratus of dual type—a dynamic and in a measure an indeterminate question, as it were—that is offered for study and decision. Independent in great measure of set mathematic rules and fixed certainties, the problem can be solved only by careful study of both the local and the general conditions, with a knowledge of how the eyes are to be employed. To do this with any degree of certainty, the premises must be made as nearly absolute and rendered as unyielding as possible. The functioning material must be put into a condition wherein the total ametropic error may be known, and as much of it is to be corrected as may be compatible with proper binocular focussing for any desired point. Were this positively obtainable without the employment of a cycoplegic, the use of the drug might be omitted, but by no means known—of either objective or subjective character—can this certainty be established in any other manner than by paralysis of the ciliary muscle.

Hunting down the imp of mischief, here as elsewhere, is a task which requires the acumen and the judgment of a broad-minded specialist who is thoroughly conversant with general symtomatology. No more here than elsewhere can the narrow-minded organologist who attacks one poor organic victim that he knows the most (or more probably the least) about, be considered of any use in the diagnosis and treatment of such a disorder. Truly, here in ametropia the special knowledge that is necessary for the recognition of such conditions should be built upon a firm superstructure that is placed on the broad substructure of general medicine.

## DETERMINATION OF AMETROPIA AND HETEROPHORIA.

Optometry, or the science of measurement of the optical power of the eye, divides itself in its practical work into two types of examination—the objective, which consists in a series of plans in which the answers are obtained independently of any response from the subject; and the subjective, which includes all those methods the results of which are offered by the patient himself.

For ascertaining the variety, the amount, and the degree of ametropia, three of the most important diagnostic methods—ophthalmoscopy, keratometry, and the fundus-reflex test—should be employed. In the estimation of the ametropia, keratometry, subjective testing with test-lenses, and the fundus-reflex test should be used. For the determination of related muscle imbalance, both objective and subjective tests should be applied not only during distant vision and for the ordinarily used working points, but for any other desired situation of monocular and binocular focussing.

Every case of refraction in an intelligent subject must be submitted to a series of final subjective tests before the many varying and the more or less certain objective ones can be accepted as conclusive. The objective tests should be considered more useful for diagnostic purposes than the subjective ones.

In the ordinary examination of the eye (not perfunctory routine, which,

at the best, is worse than useless), certain procedures should take precedence. After the history of the case has been gotten, the acuity of vision in each eye separately and with the two eyes combinedly is to be ascertained. This done, the monocular and the binocular powers and ranges of accommodation should be obtained. The sizes and the shapes of the pupils, with the action of the irides, are then to be studied.

This is to be followed by careful ophthalmoscopic, keratometric, and retinoscopic examinations. The powers, the tendencies, and the deviations of the extra-ocular muscles are then to be tried during both near and far visions. These studies, with a noting of any relevant local and general symptoms, are to be made before any opinion can be given as to the diagnosis and the treatment of the case.

At the next visit, if a cyclopegic has been used, vision for distance and extra-ocular muscle-balance for distance are to be obtained, while certainty of total cyclopegia is to be assured. These determinations are to be followed by keratometry, loose-lens selection, gotten through carefully centred and properly balanced trial-frames or brackets, and retinoscopy. This work is to be repeated daily, if possible, until two sets of certain and definite answers have been proved to be alike.

After waiting a proper time in order to be sure that the effects of the cyclopegic have disappeared, the lenses that were previously chosen are to be tried in carefully fitted trial-frames. All of the astigmatism, as a rule, and as much of the hypermetropia with as little of the myopia as may be consistent with good or the most useful vision, are to be corrected; taking care that the best muscle-balance, the nearest approach to orthophoria, and the least disturbing binocular action are all gotten.

At the last visit, the glasses and the fittings of the frames are to be measured and proved; when, if all is found correct, the patient is to be given a few words of advice as to the care of the glasses and the use of his eyes.

If the subject is not intelligent, most of the work will be limited to the objective methods.

Concise though adequate records should be kept of every relevant detail that may be of future use.

*Keratometry.*—The keratometer (so popularly and improperly known as an ophthalmometer) is an instrument which, when properly employed by a competent and normal-sighted observer, is one of the most valuable contrivances for diagnostic purposes that has been placed in the physician's possession. Single determinations with it, however, cannot be accepted as final. Stripped of all the profession that has been made for it, it resolves itself into a piece of instrumentation that is intended for the determination of irregularities of the anterior face of the cornea.

It is useful in estimating high grades of corneal astigmatism in which vision is ordinarily defective. It is valuable in determining the major and the minor meridians of the anterior surface of the cornea in some cases of

irregular astigmatism, as, for example, in the forms that are at times produced by the evanescent changes of cicatrization that follow corneal sections, and are seen during temporary pressure or tension and traction on the cornea from various causes, etc.

The fact that the main purpose of the method in ophthalmic practice is to expose meridional faults that are resident in the anterior surface of the cornea alone, proves that it is faulty even as keratometer,—it failing to take any note of the posterior surface of the membrane as well as of any irregularities that may exist in the corneal tissue itself. This is true even in the visual zone, which is the portion of the cornea that is estimated by the method, and which, although constituting a part of an ellipsoidal surface, is fairly spherical in shape.

Moreover, the amount of corneal astigmatism does not always represent the total amount of astigmatism of the eye. Instruments, such as the so-called "ophthalmometers" (keratometers) of Javal-Schiötz, Leroy and Dubois, and Reid, that measure a portion of the cornea alone and fail to give any clue to any form of intra-ocular astigmatism whatever, cannot, like the fundus reflex test for instance, be considered unequivocal in the proper determination of all the astigmatism of an ordinary eye.

The makeshift plan of empirically adding and subtracting fixed cylindrical equivalents to and from the actual readings (indeterminate quantities that in themselves can give rise to the most pronounced eye-strain) obtained by the method, is neither logical nor right. The very fact of the necessity for these hap-hazard calculations shows the fallacy of the method as determinative of the total amount of astigmatism that may be present in an eye. This is true of any keratometer, no matter how perfect the instrument may be and how carefully the technique may be applied; the slightest uncertainty and the least doubt in result will swerve the answers from—what must always be sought for in careful refraction work—absolute accuracy.

Practically, then, the newest and the most improved American models of keratometer of Javal-Schiötz, or the better-constructed instrument of Leroy and Dubois, and that of Reid—which last should always be preferred—are most useful, when carefully applied, in determining approximately the angles of the axes and the degrees of the various meridians of corneal astigmatism. The relative lengths of the radii of curvature of the cornea may give an indirect clue to the existence of an axial ametropia, the rule being that the long radius and, of necessity, the flat cornea are the concomitants of hypermetropia, while the opposite condition is associated with myopia.

*Fundus-Reflex Test.*—Retinoscopy, skiascopy, or retinal-shadow-test, as the plan is at times called, is so easy of manipulation, so certain and so broad in its answers when properly applied, and so rapid in its application, that its use should never be omitted from any case of faulty refraction that may be offered for diagnosis. It is also of value as a prover of subjective findings, and it is of use in ordering lenses for those patients for whom

subjective methods are impossible. It helps the work of subjective choice of lenses, obviating the necessity for entire dependence on the findings of the patient.

Like all other objective tests, it is valuable, though to a greater degree, in the cases of those with whom it is difficult or impossible to hold communication—the mentally or sensorially deficient. In fact, it may be usefully employed while the subject is in a somnolent condition, though, of course, in such cases, the primary position of the eyes, and hence the portions of the visual zone studied during this state, must be taken into consideration.

It considers both the amount and the refractive character of all the dioptric elements along and around the entire intra-ocular portion of the visual line. It gives answer to the correctable refraction in its entirety. In this respect it is far superior to keratometry, but, unlike ophthalmoscopy, it fails to show the condition of many of the important tissues in the interior of the eye. It is also better than the ophthalmometry of Helmholtz and Tscherning: these methods, in addition, being, as a rule, too difficult for ordinary clinical employment.

The great difficulty in bringing the fundus-reflex test into every day practice as a routine procedure has been the trouble in placing the test-lenses used during the examination properly before the eyes; much time being spent during the study of single meridians for every lens employed. This objection, however, has been in a measure overcome by various contrivances that permit much easier, more accurate, and more rapid lenticular adjustment.

By the aid of a few easily applied special adaptations, and with a little practice, the test can be used by the observer for the determination of his own fellow eye, thus rendering the plan a useful objective adjuvant in the detection and the determination of the observer's ametropia—though, of course, not of that which is situated exactly in the visual line of the eye which is being studied.

*Ophthalmoscopy.*—Although the ophthalmoscope is the only certain instrument by which ciliary-muscle spasm can be detected, yet the ophthalmoscopic reading of ametropia is necessarily uncertain and variable. In the direct method the accommodations of both the observer and the observed are so unfixed; the study of the coarsest fundus changes only are usually made in an axis and a position which do not correspond with those of the macula lutea and the visual line; and the employment, as a rule, of only the gross divisions of lens-power of spherical strength in the instrument: show how, even in the hands of trained subjects and expert examiners, the plan cannot be relied upon as scientifically correct. So, too, with the indirect method: here the distinctness of the changeable aerial image, the want of certainty of any definite planial inclination, and the, almost without exception, unmeasured or coarsely estimated situations at which the images are placed, evidence at once the fallacies that may creep into a plan in cases in which absolute facts are so necessary.

The great advantage that the method has over keratometry and the fundus-reflex test, however, in revealing the condition of some of the most important intra-ocular structures, makes its use a necessity in the study of all cases of ametropia; it giving an almost absolute answer as to the necessity or non-necessity for lens-correction.

*Loose-Lens Selection.*—Even though the entire amount of the corneal astigmatism has been obtained by the keratometer, and the character of the ametropia has been determined by the ophthalmoscope, it yet remains necessary for the employment of this variety of subjective test in the intelligent while the eye is under the influence of a cyclopegic (or its use with the fundus-reflex test in the mentally uncertain and sensorially defective) to give the best possible work to those who come for advice.

To the scientific and the conscientious physician there is no temporizing. In those cases in which the best results are expected and can be given, there should be no makeshift, no matter how slight a doubt may exist. In all therapy it is the duty of the medical practitioner to avoid any doubtful method when he has at his command one upon which he can rely with absolute certainty. Before any formula for lenses can be legitimately written, all available data that may be obtained must be sought for and gotten. Anything less which is the result of ignorance is simply an error of omission, while all of that which has been ignored by those who know better becomes a sin of commission. Every particle of the ametropia must be estimated and known before lens-therapy can be applied: the diagnosis must be completed before treatment can be attempted.

In loose-lens selection the best and the most improved practical apparatuses, with the greatest care in their adjustment, are necessary to give positive determination to each individual case. The adaptation of Scheiner's experiment to several ingenious mechanisms, by which not only the various forms of ametropia can be determined, but the different grades may be estimated, can at times be made most useful.

*Optometry without the Use of Drugs.*—From time to time, many most excellent plans have been tried, in the hope of obtaining the total amount of ametropic error without the use of cyclopegics. Each method, endeavoring to overcome the constant impulse of an acting muscle, wages, as it were, an incessant and unequal contest against an indeterminate and unseen factor. The ciliary muscle, when paralyzed by a cyclopegic, fails to exert its uncertain influences, thus permitting the total amount of ametropia to appear and thus allowing a proper correction to be made.

Some of the latest forms of refractometer, in which the so-called fogging system is used to induce the ciliary muscle to relax its action as much as possible and thus approximately obtain the total ametropia, may, at times, by reason of their readiness of adaptation, be advantageously employed as fairly certain subjective tests, or can be used in conjunction with other methods in which it is not wise to employ cyclopegics. As a rule, however, over-corrections, especially of astigmatism, are unfortunately gotten

by untrained observers (the patients themselves) who are the ones to whom the test is submitted. At times the plan may be employed to advantage in ciliary spasm, especially when the condition gives rise to faulty muscle equilibrium.

*Muscle-Testing.*—The methods of testing heterophoria are, as a rule, very imperfect. The muscular conditions should be as carefully studied as possible with the most improved forms of instrumentation and gotten as absolutely as may be (not once but repeatedly) in order to obtain averages of muscle-balance under different conditions, as is done with the ametropic error before any diagnosis is given: then, and not till then, should any general or local therapy be applied to them. The duction powers of muscle combinations that produce the various combined ocular movements must always be tested.

To obtain the amount of departure from orthophoria is just as much of a necessity as to know the absolute degree of ametropia. Both represent the total error from which the getting of a normal muscle-balance is the most possible. Unfortunately, however, the muscular ideal is at times unobtainable. It is too unstable, and hence the necessity for repeated examinations in varying ways and under differently conditioned circumstances. (For details, see pp. 167–188 in vol. ii. of this System.)

## CORRECTION OF AMETROPIA AND HETEROPHORIA.

The problem of lens-testing is different from that of lens-prescribing. Lens-testing consists in the trial of a number of objective and subjective plans in order to obtain the total amount of lens-strength that is necessary to bring a dioptric apparatus to its best possible focussing power. Lens-prescribing, on the contrary, as will be explained later, brings into consideration a whole series of dynamic forces that are situated in and around two separate and yet two closely related eyeballs; a problem, when looked at in its full force of meaning, which is one of the most difficult that any conscientious and properly taught physician can have given to him.

For the relief of eye-strain, lenses should be ordered in the same manner, for example, as drugs are prescribed. Just as, when a pulmonic disorder decreases, the dose of a therapeutic agent is changed or the form of a drug is altered, so in asthenopia, no one pair of combination lenses is to be considered as permanent, as is so frequently done by the laity, and so unfortunately concurred in by the vast number of optical mechanics.

The conditions, though unlike, are the same in the fact that there are a disordered physical material and an improper physiologic result. In each case the cause is to be searched for and treated, and the main symptoms are to be relieved as quickly as possible by appropriate means. Antispasmodics for the one and correcting lenses for the other, for example, do not directly effect a cure: they simply, when properly applied, allay harmful symptoms and thus indirectly remove one or more of the disturbing factors that are seen during the progress of the disease.

Lens-prescribing, thus legitimately given to the medical man, becomes one of the most important therapeutic agents that has been offered to the profession. Just as a drug affords rest and resolution, just as a splint immoblizes the broken parts and permits restitution, and just as a brace removes inequalities and gives opportunity for performance of correct physiologic acts, so, here, lens-therapy serves its part.

In all lens-prescribing for myopia as well as for hypermetropia, the best possible associated correction of the two eyes both as regards refractive error and binocular fixation, is to be sought for, after the estimation of the total amount of the refractive condition has been made in each eye separately. This done, comfortable and sustained combined vision with the two eyes can be generally obtained. If not, exercise, orthoptic training, hygiene, modifications of lens-power, attention to the general health, and, lastly, radical interference with the lengths and the relative positions of the extra-ocular muscles in order to obtain new and better-balanced muscle-equilibrium, may all be tried. In brief, no case of binocular ametropia can be considered as having been judiciously and properly treated until the disturbances of both the related extra-ocular and the intra-ocular muscle-groupings, which are brought into almost momentary play, are gotten rid of as much as possible.

Severe eye-strain, although marked in itself, may be productive of apparently unrelated and yet seemingly grave symptoms to such a degree as to mislead ignorant and careless observers into the belief of more deeply seated and more dangerous conditions. It is in just this type of cases—those especially of the minor varieties of asymmetrical astigmatism and low degrees of unequal hypermetropia with good vision—that the seemingly sudden reliefs and the most remarkable cures from distant distressing symptoms are obtained.

The greatest judgment and the utmost skill are necessary in the prescribing of lenses for any pair of eyes. So much is to be taken into consideration, and such slight modifications are so productive of such harmful results, that a medical education and a thorough training in this special form of work are obligatory. This is better understood when it is considered, for instance, that the production of an artificial ametropia, as it were, is not infrequently necessary in order to obtain a pair of comfortably working and unirritating visual organs.

An emmetropia with an orthophoric or perfect muscle-balance which is different from a normal balance may not be the ideal state of two eyes that are working together, as the two conditions may be inconsistent with harmony of action. The association of a mathematically correct optical constant with a supposedly perfect equilibrium of dynamic forces is almost an impossibility: but one set of conditions is fixed.

Practically, it is the best interrelational condition that is necessary for comfort and structural integrity that should be gotten. Such a condition may necessitate a departure from emmetropia to bring the related muscular

apparatus into a normal balance and an easy working power—a relation that may require frequent change before a comfortable and harmless standard is obtained. Fortunately, the more or less fixed dioptric premises being obtained and corrected, the changeable ones are apt to fall into their least disturbing balance, necessitating less help to give them a normal working power than before the ametropia was removed. The secret is, to bring about as close a proper relation between the two conditions in each individual case as possible, hoping only to approach the ideal state.

The full correction of the meridional defect with as much of the axial error as possible will not infrequently, when combined with the correction of a heterophoria (particularly a hyperphoria, which will often cause much of a faulty lateral tendency to disappear), give the desired result of a pair of normally working eyes. In hypermetropia in which the ciliary muscles have just recovered from their artificial paralyses, more of the combined hypermetropic error will be borne during binocular vision than during monocular vision.

As a rule, the careful and the as nearly as possible total correction of unequal degrees of binocular ametropia (particularly the minor types) not only removes a false tendency of the globes in some definite directions, but does more—it restores both the extra-ocular and the intra-ocular series of muscles to a normal interrelational balance, thus permitting easy and proper equipoise without undue or harmful innervation impulses to take place when the two globes are moved into any associated and incident positions.

When there is a tendency to inward deviation remaining after a full correction of astigmatism with a partial correction of hypermetropia has been made, the two eyes will be brought into better muscular association, and, as a result, will act together more comfortably, the more nearly the total amount of the hypermetropic error is reached. When there is no outward tendency in the same character of eyes, much less of the hypermetropic correction can be borne with a corresponding degree of ease and comfort.

On the contrary, if there be a normal muscle equilibrium or a tendency to outward deviation in myopia, it will be found that a much higher degree of correction can be employed with safety and comfort, after the correction for the astigmatism has been placed, than when there is a tendency to an inward deviation of the two organs.

As a rule, if comfortable balance does not take place after a sufficient time has elapsed and the general health is in good condition, it becomes quite certain that there is some inherent inability in the muscles themselves which needs radical treatment: then, and not till then, should operative procedures be resorted to.

In brief, all ametropic correction, particularly that with convex cylindrical lenses, causes not only heterotropic but also heterophoric conditions to disappear, by placing all the related ocular muscle-groupings of the two eyes within as near as may be a normal and undisturbing balance for what-

ever working points the lenticular combinations that have been applied are intended. This is obtained not only by the visual improvement of each eye alone and of the two eyes combinedly, but also by the restoration of harmonious and correct binocular focussing at the habitually used points of individual election: an artificial harmonious innervation of the entire extra-ocular and intra-ocular muscle-groupings which is adapted to the corrected ametropia, no matter what its type, its amount, or its degree may be.

If two eyes with, for example, two diopters each of uncorrected axial hypermetropia or myopia, can do their work better, with more comfort, and with greater safety to their physical welfare than if fully corrected, then that amount of corrected ametropia constitutes the axial normality of that individual pair of eyes, and that must be the therapeutic plane to be sought for in that particular instance. If two eyes need over-corrections or if they even require unequal corrections of axial ametropia to restore binocular vision with the least amount of motor and sensory wear and tear, then such a pair of over-corrected or unequally corrected eyes is better conditioned and more scientifically cared for than if each ametropic eye had been gotten to an axial emmetropia with a resultant inequality of binocular action. No law in these classes of cases can be made to serve each individual instance. The premises are too unstable to allow the formulation of any fixed rules in every case. The empiricism of common sense, good judgment, and a knowledge of the requirements of each case, based on scientific principles and sought-for details, are the only means that can be at all relied upon in ordinary practice. In all corrections, therefore, emmetropia should be sought for as nearly as possible, but, above all things, as close a normal relationship between the two eyes as can be gotten must be established.

In the majority of cases the question is to get the two eyes to work harmoniously together for the generally employed distant and near working points. These two positions are the most important during the combined use of the eyes. The problem is to reduce the optical and the muscular strains to a minimum in agreement with what is expected of the organs to perform. Normality and emmetropia need not be the same. As can now be understood, the mathematical lens-basis upon which emmetropia is assumed may not be the best practical basis upon which to place the two organs.[1] This can be readily appreciated when the over-developed ciliary muscles of hypermetropia, with their too great influence on the play and interplay of the exterior ocular muscle mechanism, are contrasted with the influence of the weak and little-used ciliary muscles of myopia upon their extra-ocular muscle series.

In cases of heterotropia in which monocular amblyopia is threatened,

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[1] That portion of orthophoric imbalance (heterophoria) which is dependent on other causes than ametropia and is not benefited by correction of the ametropic condition, although germane to the subject, will not be considered in this article, as it belongs to other chapters in the work.

correction of the ametropia, stereoscopic exercises, and periodic use of the badly functioning organ should all be carefully and prolongedly tried as early in life as possible. If in this type of case it is found that the vision of the affected organ remains at all below normal, immediate resort should be had to radical procedures. Each case, however, should be made to serve as its own precedent, with remembrance of the all-important fact that every good working eye is of inestimable value and that a pair of such eyes is priceless.

Although radical procedure does its work much more rapidly than the use of prisms, yet it must not be forgotten that each form of therapy has its limitations and should be judiciously used. Operation should always be considered as an ultimation in the therapeutics of ametropia, heterophoria, and heterotropia.

While the orthopædic treatment of ametropia may be but one of the methods that is necessary to be used not only for the removal of the etiologic factors at work in the establishment of the complexus of asthenopic symptoms in any given case, but also for their recognition, yet, as a rule, it really often is, and should be frequently so considered. For instance, differentiation should always be attempted between a cephalalgia that is solely dependent on ocular strain, and which is relieved by lenses and prisms, and one that is due to other and more wide-spread neuropathic changes, and which cannot be so remedied.

In many cases it is certain that the use of prisms in spherical combinations instead of cylinders of no matter how weak a strength signifies uncorrected muscle-balance that might have been remedied by the use of proper sphero-cylinder lenses.

*Special Treatment of Idiopathic Heterophoria.*—The only type of heterophoric asthenopia that can legitimately permit of operative interference is the idiopathic. The fault, if it is to be corrected by surgical procedure, must be located in the part upon which the operation is to be done. The causal trouble must be strictly an anatomic one. It must be "structural" or "insertional" in character.

For the removal of reflex or relational disturbances search must be made for the reflex irritant or the offending relationship. This is as certain and as unimpeachable as an absolute dogmatism.

In the idiopathic type of heterophoria in which there is some gross fault in the anatomic formation of the exterior muscles of the eyeball, operation for the relief of the defect is the only method. In this class of cases the results of a radical procedure are as sure as the abnormal anatomic relations are understood and the proper technique has been pursued. The fault should not be one of improper innervation from a central or a peripheral cause. The error should not belong to the functional type of disorder. It is a local physical deformation with an imperfect and disturbing physiologic result. Orthopædic removal of any existent ametropia by artificial lens action, improvement of general tone, and graded muscle-exercise,

either through the ciliary muscles by lenses or through the exterior muscles by decentration and prisms, are of no value. Anatomic readjustment of improperly placed muscular tissue and tendon is the only legitimate resort.

Whatever radical measures for the shortening of tendinous attachments in idiopathic heterotropia are necessary, care should be taken to confine the procedure to the tendon of those muscles which are the most prominently exercised during the more important acts, and, if possible, always to associate an advancement of the antagonists. The operation, however, need not, as is so strenuously urged by many, be carried to orthophoria. It must be gauged, if possible, and repeated sufficiently often, if necessary, so as to carry the general balance of the combined ocular-muscle series to the best equilibrium and to obtain the nearest to perfect action for the most commonly employed working-points.

If an exophoria remains after the proper correction of an ametropia, gymnastic exercises with carefully constructed stereoscopes are so useful that they can be advantageously added to the ordinary therapy that may be used. Esophorias of less than two degrees are often corrected by decentration. Higher degrees frequently require the temporary use of weak prisms, and in some instances—though not in many—they may necessitate radical procedure.

*Special Treatment of Functional Heterophoria.*—In the functional forms of heterophoria left after careful corrections of ametropic error, and in which, as a rule, the general system is below par, and thus places the local conditions in a similar state, much good can be accomplished by attention paid to the general nerve-tone with local and systemic hygiene. In this class of cases, as the subject betters in general health, rhythmic exercises not carried to excess, and in some instances innervation-impulse tests, are of use. Tenotomies made on this class of subjects are worse than useless.

These two types of heterophoria are as distinctly separated as can be, though both need the special skill that is necessary for the employment of the methods which are best able to bring the peculiar symptomatologic groupings of each into evidence, with the power to apply proper therapeutic measures to them; two procedures that can be accomplished only by the scientific, the well-grounded, and the practical medical man.

To reach such a case judiciously the ametropia in each eye must be ascertained in its entirety, and lenticular combinations gotten that are able to produce as near normal vision in the two eyes as possible with a binocular working equilibrium. Superadded to this comes the employment of the various methods of decentration, prisms, rhythmic exercise, innervation-stimulus, and general treatment, either alone or in association, as the peculiarities of each case demand. At times, and not rarely too, latent ametropia may, even while giving rise to heterophoric disturbances, remain so concealed to the gross ophthalmic procedures that are ordinarily instituted for its detection, that it is denied by the patient, and may remain unrecognized by the physician unless it is properly exposed by repeated study.

If the external muscle-groupings are suffering from secondary exhaustion, much good to obtain and to retain a normal working balance between the two related series may be accomplished, in many not too pronounced cases, by carefully chosen exercises with the innervation-impulse tests. Never, however, in either heterophoric or heterotropic errors should attempts be made to remedy abnormal muscle disturbance by weakening any set or sets of muscles. Endeavor should always be made to find, if possible, the weak series, and to strengthen them, either indirectly by removing the ametropic strain, or directly by orthopædic measures (or even by radical ones if absolutely necessary) on as much of the entire muscle-grouping as may be deemed proper in each instance.

Some cases of heterophoric asthenopia of the paretic type will persist in spite of the most careful ametropic correction, with apparent restoration of binocular fusion to orthophoria for both near and far visions. In this series the unrelieved condition is evidently dependent on general systemic disturbance. Here, although the volitional centres may be at fault, both systematic hygiene and internal medication are indicated. Empirically in such cases, if the muscle-equilibrium be such that an exophoria or possibly a hyperophoria as a part of a functional paresis of the third nerve be present, the best results can be obtained by full doses of nux vomica and strychnine administered hypodermatically. The internal administration of cannabis indica in some of these cases has been favorably spoken of. Should, on the contrary, an esophoria be found, ascending doses of hyoscyamus given internally, with the local employment of cyclopegics, are said to produce at least temporary betterment.

These are the types that the most careful corrections help but in a measure and are so troublesome to those who do not recognize the real significance of the symptom-groupings.

No such case can be cured by attacking the ocular conditions alone. Riddance of as much of the visual disturbances as possible by the orthopædic methods of those who are skilled in the treatment of diseases and disorders of the eye, with betterment of the general nerve-health in every instance, is the only legitimate way by which the cause in such cases can be reached and the patient can be cured: in this class of cases the former form of therapy is useless without the latter.

Frequently among the neurasthenic types of asthenopia as particularly seen among the intellectual and unfortunately sedentary classes (the over-worked and under-rested brain-workers of both sexes), optical and musculo-dynamic corrections are of but little or of no permanent value, while radical procedures on the muscular apparatus are injurious. In this class of cases prolonged and absolute change, thorough and enforced rest, and wisely chosen and properly taken exercise, both physical and mental, can alone effect a cure: here, as before stated, the ocular symptoms are but a part of the general disturbance, and can be reached and met only by attention that is given to the main cause.

*Special Rules Governing the Correction of Ametropia and Heterophoria.*—In many cases, especially in those subjects who have reached middle life before wearing correcting lenses, the erring extra-ocular muscle habits require varying periods of time before they can be overcome, even when the combined muscle series is apparently restored to normal balance by ametropic correction, decentration, prisms, and radical interference. This is probably due to a long-continued perverted physiologic act begetting abnormal peculiarity of muscle tissue, just as the ciliary muscle is found to be overdeveloped in so many cases of uncorrected and undercorrected hypermetropia.

So too with the ciliary muscles of a pair of hypermetropic eyes that have been overtaxed servants almost since the beginning of the individual's existence.

On the contrary, myopia, practically having no disturbing ciliary muscle action, immediately hails the newly found world with almost childish delight or lives anew what it may have known in the past.

In some subjects the distortion in corrected astigmatism, especially in cases in which there are oblique and asymmetrical axes in the cylindrical lenses, is so great that objects appear twisted, producing all manner of temporary anomalies of muscle-equilibrium.

In many cases of masked astigmatism, the primary resultant vision which is obtained from the use of correcting cylindrical lenses, although from the first superior to the previously or present uncorrected vision, gradually improves, this frequently taking place the earlier in the eye that has lesser ametropia.

Empirically, it may at times be necessary to give but a partial correction of astigmatism (especially the hypermetropic variety), and gradually to increase the degree of the cylinder-strength until the full amount that was found to be present during the cyclopegic examination has been reached. This, almost without exception, is dependent on an interrelation of the two eyes in their combined acts, and is rarely seen in monocular subjects.

In some cases, especially in old subjects, it will be found necessary for comfort's sake even to omit the use of the cylindrical corrections. In others, particularly in cases in which there is much astigmatism and where the principal meridians are obliquely placed, the degrees of the cylinder-strengths must be lessened and the angles of the cylinder-axes must be changed during near work. In a few of these cases the so-called ellipsoidal lens may be of value.

In some cases of high degrees of myopia in which lenses are badly borne, so-called perforated spectacles, consisting of blackened opaque disks that contain a number of geometrically placed perforations, may be of use. For amblyopes wearing high corrections and for cataract patients a rectangular black disk containing an aperture that is sufficiently wide to include a couple of lines of brevier type—known as a "typoscope"—may be of value.

It may be laid down, however, as a broad rule that, no matter what the

post-cyclopegic axis of astigmatism may be, preference should always be given to that which has been obtained when the ciliary muscle was at rest, the probable lenticular astigmatism (the main cause) most frequently gradually diminishing as the ciliary muscle regains its normal action.

In a few extremely rare cases the post-cyclopegic axis must be the permanent one that is used. This form of disturbance depends either on the selection or on the preferment of some other axes than the principal ones when the pupil has become of normal size, or is the result of an abnormal anatomic construction or pathologic change which is situated somewhere in the lenticulo-ciliary structures.

The best rule in prescribing for compound hypermetropic astigmatism is, without regard to slight degrees of fogging, to endeavor to order correction for all of the astigmatism in combination with as much as possible for the hypermetropia that will keep the muscle-balance of the combined two eyes in a state of equilibrium for both distant vision and near work, especially the latter.

If a single correction which gives good vision and which restores muscle-balance for near and far cannot be given, an additional pair of glasses for close work should be ordered, even if such a pair of glasses means a mere decentration of the lenses already used, or consists in the addition of a prism or a combination of prisms to the correction for distance. It must be remembered, however, that there is no more of a rule here to offer than there would be to attempt to formulate a law for the regulation of any character of therapy that might be addressed to any other organ of dynamic construction. Each case must be treated by itself, just as it is necessary, for example, to make a difference in the treatment of a pneumonia in the weak and of pneumonia in the sthenic.

As a rule, in all cases in which there is a slight esophoria for distance, it is best not to prescribe prisms for the correction of such a defect. In cases of low degrees of exophoria, during near vision weak prisms with their bases placed inward can be put in the correction before the helping eye (generally the left one), as they will frequently, for a time at least, produce muscular equilibrium and comfortable action. If later the muscle equilibrium be again disturbed, as it frequently is when constant near work is undertaken, hygiene of eye-employment, with temporary cessation from all application of the eyes, care of the general condition, nerve tonics, vascular stimuli, readjustment of spherical and prismatic corrections, and local exercises, with an out-of-door life, and, as a last resort, radical procedures performed on the extra-ocular muscle-groupings (especially those that disturb the vertical movements of the eyes), all may be required. In some cases in which neurasthenia and hysteria are present, rest-cure, forced feeding, and suggestion may be necessary for employment before the local changes will pass away.

On the other hand, carefully adapted correcting lenses and prisms for hypermetropia, astigmatism, and imperfect muscle-balance frequently not

only serve to relieve the strain that may be brought to bear upon a pair of feebly working eyes of the weak and the convalescent, but may often indirectly act therapeutically upon the system, and be discarded when the patient's general health is made better. Care should be taken, however, not to set aside artificial lens-power from any but firmly formed eyeballs, and to be sure to await the cessation of any general disorder or local inflammatory change, particularly in cases of myopia.

In young subjects with hypermetropia it is folly, when it is possible to place the correction of the total amount of error in position (so as to produce a proper equilibrium between the two eyes and to give their separate visual acts a unified good result), to attempt to undercorrect the eyes, in the belief that full correction tends to disturb the supposed normal outgrowth of the ametropic defect.

At times it may be of advantage to allow a patient to make temporary use of the ordinary test-lenses, placed in any desired numbers or positions in specially adapted frames, before the permanent combinations of strengths are ordered to be worn.

For several reasons, no corrections should be ordered while the accommodation is suspended and the pupillary area is enlarged. At this time, although the full amount of ametropia in each eye may be known, yet there is no knowledge, except that which is indirectly obtained during the preliminary examination, as to the balance of the intra-ocular and the extra-ocular muscles in binocular vision during work. As before stated, the question is not simply how to obtain the kind and the degree of the optical error in each eye, but it is the more complicated problem to know how much of the ametropia in each eye it may be necessary to correct when the two eyes are placed in physiologic combination. Again, pupillary dilatation disturbs the exact value of the visual functions as ordinarily gotten while the pupil is in its normal state, the rule being that the larger the pupillary opening the more uncertain are the findings. This is well exemplified, for example, during the examination of celestial bodies, in which the resolving power of the spectroscope is in direct proportion to the smallness of the pupillary area.

It must also be remembered that cyclopegics are to be used not only to aid in the getting of the proper premises upon which to formulate the kinds and the degrees of optical errors, but also as therapeutic agents in fixing, as it were, the dynamic structures of irritated and at times inflamed eyeballs, just as a broken bone is splinted and a gaping wound is coapted and bandaged. The cyclopegic thus employed reduces irritation and allows healing of inflamed tissues to proceed more uninterruptedly. In some cases of this type the permanent use of correcting lenses should be avoided until all gross inflammatory signs have ceased.

Sleep and exercise are wonderful curative agents for asthenopia. A few additional hours daily of sleep often allow a patient to get rid of much eye-strain that lenses, prisms, and tenotomies can never fully reach, while super-

added healthy and legitimate out-of-door exercise becomes a boon to both the sufferer and his adviser.

From all this it will be seen that, although the theoretic cure of asthenopia in any pair of eyes is the artificial production of emmetropia and orthophoria, yet practically such ideals, though at times apparently obtained, are rarely, if ever, preserved. The mathematic and the geometric bases upon which the dioptric formulæ are placed are uncertain. The ocular tissues, especially in the young, are yielding and extensible. The supposed optical constants are so inconstant; the harmonious relationship of the two organs, which are frequently different in their construction, is so unharmonious; and the dependence of the visual apparatus on the general health is so important, that nothing more can be expected in the removal of asthenopic conditions than to approach as near a refractive and as close a muscular ideal as possible, taking care at the same time to preserve the two organs in as healthy and as normal a condition as may be consistent with their proper combined physiologic powers.

*Special Treatment of Spasm of Accommodation.*—In the treatment of ciliary spasm, careful alternating use of cyclopegics and mioties, the employment of smoke-tinted coquilles that are either plain or are made to correct the spastic disturbance, with general hygiene and the employment of nerve stimuli and digestive aids, are all of value. The sooner that the proper lenses for the correction of the ametropic error can be worn in such cases, especially those for the correction of the astigmatism, the more certain it will be that the spasmodic state of the ciliary muscles will gradually disappear. All manner of temporary changes of lens strengths may, in accordance with the necessities of the case at the time, be tried to advantage and without detriment. Orthoscopic and prismatic exercises, in order to relieve any over-stimulation of the internal rectus muscles, may often be usefully resorted to.

Some cases of spasm of accommodation, however, are so persistent in type as to require months of active treatment (both local and general) before the true grade of refractive error can be arrived at.

*Special Treatment of Megrim (Migraine).*—In many instances megrim has one of its main causes in ocular disturbance, and can frequently be benefited by attention that is directed towards the visual apparatus. In such cases it is doubtful whether bromides given between the exacerbations are of any value. Clinically it seems much better to get the eyes into a normal state and hygienically to treat the generally associated neurotic condition, which is also probably one of the principal causal factors in the production of the affection.

*Special Treatment of Habit-Chorea.*—Typical habit-chorea of refractive origin, like any other similar type of choreiform movement, has a tendency to become increasingly uncontrollable and to invade other muscular groupings besides those of the eyelids and upper face. Such cases, though controlled in a measure by constant mental effort, which frequently means

undue vigilance and at times painful strain, are bettered by corrections of ametropia and heterophoria, which may set the mimicking clonic spasms of the affected muscles temporarily at rest. The condition, if at all times pronounced, can be cured only by additional therapy that is addressed to the welfare of the system as a whole. In many cases the transfer of the patient from a sedentary life to an active out-of-door occupation will be of the greatest benefit. Weak solutions of eserine or of any drug of like nature, with the employment of overcorrecting convex lenses, may at times be of therapeutic advantage. Study of the nasal mucous surfaces should always be made, and attention should also be directed towards the possibility of osseous deformities in the upper portions of the nose. Antral disorder arising, as a rule, from carious teeth should be thought of. The possibility of the existence of more distant causes, such as uterine and pelvic disorders, particularly in young adults and even the more mature, must never be forgotten; and, lastly, the great army of so-called neurasthenics, gathered from every locality and social condition in this too-fast-running age of ours, should not be lost sight of. If to the results of these studies is combined carefully graded internal medication with arsenic, or, better, hypodermatic doses of the same drug, a cure can nearly always be gotten.

*Special Treatment of Cephalalgia.*—Among the writings of the older authors on general medicine, and in some of the works of the present time, expressions may be found in which an inability to cure constant headache by ordinary medical therapy has given rise to the consoling thought that the neuralgia will disappear spontaneously as the age of the patient advances. In fact, examples of these types of cases are now of no rare occurrence in ordinary office practice. How many of them might have escaped their distressing and almost life-long attacks of cephalalgia or other related neuroses if careful study of their visual apparatuses could only have been made early in life, it is impossible to know. It is presumable, however, that a goodly proportion were victims of unrecognized low degrees of ametropia, especially of the hypermetropic and astigmatic varieties.

In reality, much of the cephalalgia that was formerly treated by blood-letting, emesis, and purgation owed a great deal of the temporary relief which was obtained by these means not only to the exsanguination and the removal of disturbing material from the digestive tract, but also to the enforced rest from near work that was given to the ocular apparatus itself during the exacerbations.

From all this it can be understood that, to be on the safe side, it would be well to estimate the refraction in all cases of functional neurosis of this type, particularly that which is situated in and around the eyes.

*Special Treatment of Lid-Inflammation.*—Blepharitis, especially when of squamous type, may often be benefited and even cured by careful correction of ametropia, with or without the topical application of such ointments as the yellow oxide of mercury or resorcin with sulphur. During the use of the latter ointment care should be taken to remove the scabs or

sycotic masses that are found at the hair-roots by the use of stupes of hot solutions of boracic acid, table salt, or bicarbonate of sodium. In the graver forms of the disease applications of nitrate of silver or of solutions of carbolic acid, made by the physician, are invaluable.

*Special Treatment of Presbyopia.*—Almost without exception, it is folly to attempt to estimate a presbyopic error without first carefully determining the ametropia of each eye separately by loose-lens selection, without a cycloplegic if possible, and taking it into consideration. This done, the artificial combined points for near work that are useful to the subject (be they one or more) can be readily obtained by further loose-lens selection. The same care to see that everything is properly centred, etc., should be taken in this class of cases as with the younger subjects, who present the most disturbing of symptoms.

The "lorgnette" (lorgnon) which is so often used is so impracticable, so uncertain in its employment, and so difficult of proper adjustment, that whenever possible it should be discountenanced by those who have the guardianship of the human eye. In a few cases, such as simple hypermetropia, myopia, or presbyopia, without astigmatism or disturbed muscle-equilibrium, a carefully fitted pair of spherical lenses in such a style of hand-frame, that can be employed for momentary use for either near or far vision, may be ordered.

*Spectacles and Eyeglasses.*—The making and the correct fitting of spectacle-lenses before the eyes are just as necessary for proper result as the selection and the ordering of the lenses themselves.

The patience which has been exercised and the skill which has been used by the physician in order, by every subjective and objective method that he has found necessary in each case, to obtain a knowledge of the ametropia in its entirety, and thus assure himself how much and what portion of it he is to employ in the correction-lenses, are well counterbalanced by the care which must be taken by the optician to see that the lenses that have been ordered are correct and that they are placed in accurately fitting frames. The fulfilment of these conditions and the giving to the patients of the strictest injunctions in regard to the use of the lenses, the care of the frames, and their periodic readjustment, are of greater weight in the prevention of increase of ametropia, especially the axial and meridional forms, than is ordinarily imagined.

Proper correcting lenses, placed in substantially made and adequately fitting frames that are periodically readjusted, do much to retain the primary degree of refraction for which the lenses were obtained. This is true of both hypermetropia and myopia, particularly in cases in which there is some astigmatism present. These are the cases that, as a rule, not having at first the apparently beneficial effect of increased vision given to them, too readily learn to either neglect or throw aside valuable material that to them does not seemingly give what is so popularly and so unfortunately expected —mere betterment of vision. These are the cases in which false judgment

and bad decision could have been avoided had the patients been taught that there is a strict orthopædic value attached to every properly chosen lens, both in regard to its giving ease of function and to its ensuring safety and comfort to a corrected organ.

Spectacle-frames should be made of "fourteen-karats-fine" gold or of well-nickelled or blued steel. They should be rigid, firm, and durable, and yet so constructed as to be neat and inconspicuous. The greater part of the bridge-length should fit upon that portion of the nose upon which the weight of the frame will naturally fall. The frames should be free from vibration. All of the fixed joints, particularly those of the bridge, the strips, and the end-pieces, should be immovable. If the spectacles or eyeglasses be what is known as "frameless," in which without binding wires the bridge straps are bolted or soldered directly into holes in the lenses themselves, they should be secure, firm, and rigid. The temples should fit the temple inequalities as much as practicable and extend directly back to the upper junctions of the auricles, at which places they should curve down properly behind the ears. Every part of the mechanism must be made to conform correctly, thus frequently necessitating the two halves of the frame to be made unlike.

As regards the lenses themselves, the best material, without any flaws, imperfections, or blemishes, should be employed. Unless otherwise ordered, the optical centres of the lenses that are to be used for distance should coincide with the pupillary centres when the eyes are made to gaze at a distance. When near-lenses are ordered, their optical centres should be obtained while the eyes are fixed upon the specified near-point that has been chosen, and the planes of the lenses should be inclined at a proper angle.

In every case care should be taken to prevent the eyelashes from striking the back surfaces of the lenses. Spherical lenses of low and medium grades of strength are ordinarily made meniscus in shape, the concave surface being situated towards the wearer's face. When higher strengths are used, the double similarly surfaced (biconcave and biconvex) forms are employed. In the compound forms of sphero-cylindrical lenses, with a few rare exceptions, the less projecting surfaces should be situated towards the eyes.

In quite a number of instances various forms of bifocal lenses can be employed successfully, saving much time and trouble to the wearer after his disturbing probation has ceased. The most frequently used inferior segment, of such shape and size as may be required in the particular case, is useful for near-wear, while the upper segment in near-work lenses, through which momentary distant glances can be had—for example, by clerks, stenographers, etc., while engaged in near work—is peculiarly valuable. In every instance experiments in regard to the size, the shape, the centring, and the kind of bifocal segments should be repeated sufficiently often to obtain the best possible results in each given case.

The unfortunately frequent habit of carefully corrected myopes of ignoring their near-work lenses, either through laziness or from a desire to be able momentarily to see distant objects without the necessity of changing lenses while occupied in close work, should be deprecated. The use of a segment in the upper parts of the reading lenses of such subjects will often be sufficient to break them of this injurious habit.

In some of the more recent forms of bifocal lenses, the materials in the two areas are constructed respectively of crown and of flint glass, the difference of density between these two substances giving in some cases quite a difference of focus with almost the same arc of curvature. More recently the stronger lenticular segment has been buried, as it were, in the substance of the weaker lens, and thus practically rendered invisible.

In the lens-correction of aphakia, a doubtful advantage has been gained by superimposing over each of the reading lenses a concave hinged lens of a strength that is relatively equal to the difference between the chosen reading and distance lenses, thus allowing the wearer the opportunity of swinging the concave glasses out of position when he desires to employ his eyes for near work; but there is no real necessity for such a clumsy contrivance while bifocal lenses are constantly being employed most successfully in such cases.

To obviate the greater part of the aberration caused by the strong spherical lenses needed in many cases of high hypermetropia,—as, for example, in some cases of aphakia,—the lens-combinations may be so made with crown and flint glass that the visual field becomes practically aplanatic.

The plan of having several lens-strengths for differently employed foci is not new, however, having been a regularly pursued and constantly applied method for a long period of time among practical workers and wearers.

Could the correcting lenses be made of more pliable material and with adaptable foci, instead of rigid glass with only one focus, ametropic measurement would not be restricted to the determination of fixed points. It is not improbable, however, that combinations of adjustable spectacle-lenses might be constructed that could be regulated by the wearer for any desired point of view.

In the placing of prisms before the eyes in lenticular corrections, it is best to put the bulk of the prismatic action before the helping eye, which is generally the left one. If there be but a minor degree of heterophoria and but a weak prism is to be employed, the entire prism-value may generally be better placed in front of the same eye.

In order to correct irregular corneal astigmatism, small glass shells, or lenses with their anterior faces made with curvatures that are equal to those of the normal cornea, have been tried. They are so arranged that their posterior surfaces can be placed almost in contact with the cornea, the intervening space being filled with a sterilized solution of grape sugar. As the result of attempts that have been made upon man and upon some of the domestic lower animals to ascertain the periods of time that these shells can

be borne without discomfort or injury, it has been found that the subjects experimented upon were able to wear such lenses for several hours' time without their experiencing any inconvenience from clouding or irritation.

*Verification of Spectacle and Eyeglass Lenses and Prisms.*—As all the formulæ for lenses and prisms are capable of being verified by the physician, every combination, no matter how simple it may be, should be carefully proved in every detail before it is allowed to be worn or to be used by the patient.

Lens surface measures of various kinds not only are of great value in determining the exact amounts of curvatures of concave and convex spherical lenses, but with special adaptations may as well be employed for the accurate determination of cylindrical surfaces. The strengths of periscopic and parabolic lenses, crossed cylinders, and toric lenses can be easily estimated, with the additional advantage that the exact nature of each surface may be definitely determined.

Contrivances which ordinarily are nothing but adaptations of gauged protractors are useful at times in estimating the degrees of the opening angles of prisms, and thus approximately giving their deviation-values.

One of the best methods to determine the position of the cylinder-axis in a spectacle or test-frame lens is to gaze through the lens at a card containing a series of divergent lines radiating from one another at angular differences of five or ten degrees each, placed at double the distance of the lens-strength away from the eye, care being taken accurately to centre the lens and the test-card.

*Care of Spectacles and Eyeglasses.*—The lenses should be kept scrupulously clean and the frames should be handled carefully. It is highly desirable that neither spectacles nor eyeglasses should ever be folded: in fact, those that are rigid throughout are much the best. When the glasses are not in use they should, if practicable, be placed on a flat surface (preferably a convenient shelf made for the purpose) in some particularly chosen place.

*Discomforts from the Use of Lenses and Prisms.*—The metamorphoses that are produced by correcting lenses should be fought against as much as possible by the patient. The undue habits should be conquered as quickly as may be consistent with useful vision.

Too pronounced distortion of images may be compromised for in a measure by less disturbing corrections; but these should be considered merely as temporary, and carried carefully forward as rapidly as possible until complete and total combinations can be obtained. Every pair of cylindrical lenses of no matter how slight a power must furnish its quota of disturbing prismatic influences, and, when of high strength or when the axes are obliquely or asymmetrically placed, can be successfully worn only after the most patient and the most persistent trial. In apparent contra-distinction, but for similar reasons, when the subject's eyes have been brought to a proper refractive state and the corrections have been constantly worn

for some time, corresponding metamorphoses will appear when the same previously disturbing lenses are temporarily removed.

*Surgical Treatment of Ametropia.*—Excluding cases of ametropia in which proper correction of the refractive error (especially of the astigmatism) has been made in each eye, cases in which judgment as to the needs of the patient has been exercised in the selection and the fitting of the lens-combinations to be worn, and cases in which judicious care as to both local and general hygiene has given useful working eyes and has kept the disease in abeyance, there will remain but a few rare types in which any form of radical procedure is of value.

The chief reason given by those who advocate operative measures in so many cases of high-grade myopia—the inability of patients comfortably or even judiciously to wear full corrections—is not, practically, a valid one, as will be borne out by the experience of all investigators who have had considerable clinical work and have learned to adapt the strengths of the lenses that are to be used to the special wants of each case, taking care, whenever possible, to prescribe the full cylindrical correction.

That the plan, old as it is (at least more than one hundred years), may have its legitimate field, though a very limited one, there can be no doubt. Young adults are the best subjects for operative treatment. In cases in which other legitimate orthopædic plans seem useless,—as, for example, when the bulk of the myopia is lenticular in type, or particularly when the transparency of the lens is involved or its zonule is partially broken,—its removal is justifiable. In other cases the procedure is more or less uncertain.

Fortunately, here, in the United States of America, preventive medicine has done so much to lessen myopia that there are very few cases requiring any such plan of operative procedure.

The amount of refractive error left after the operation varies considerably, it never being so low as is generally taught. The fact that it ordinarily takes a myopic eye of about seventeen or eighteen diopters grade to have its refractive media reduced in dioptric power to emmetropia by the removal of the lens can be understood when it is realized that the original diopter strength of the eye has been calculated while the crystalline lens was in its normal position, and not from the position of the glass lens that is placed in the air about fifteen millimetres in front of the organ. (The writer has had one case in which there was a difference of twenty-six diopters.)

The operation, which may consist in discission alone, discission with curettement, or rarely extraction, should be done preferably on eyes that fail to evidence any gross organic disease. If possible, the posterior capsule of the lens should be undisturbed. The vitreous humor should be left intact, as its loss during the removal of the crystalline lens is of very grave prognostic import, the vitreous body being an organic element which, when once disturbed by faulty lymph-stream circulation or by mechanical influ-

ences from the products of low-grade inflammation, becomes sooner or later so disorganized as to be rendered harmful in its influence upon the organ in its entirety.

As there is usually an asymmetry of corneal curvature in such cases, producing, as a rule, a regular form of astigmatism (just as in other forms of ametropia), advantage, it is said, may be taken at times, from the readings of an ordinary keratometer, to place the corneal incision in such a position as afterwards to lessen the astigmatic error.

Operative procedure on the one eye, awaiting a sufficient time to determine the final results on the organ, should always be made before the fellow eyeball is touched.

Should signs of secondary glaucoma appear after the operation has been completed, the use of miotics or the performance of a broad peripheral iridectomy will often become imperative.

Probably the ideal result would be that of leaving about two diopters of myopia without any astigmatism, by which the patient, if content with fairly good distant vision, would have the advantage of not requiring any correcting lens during ordinary near work.

That there has been an increase of myopia after the procedure has been authoritatively stated. It may be, and in fact it is most probable, that in all cases of acquired aphakia in youth and young adult life, the ciliary muscle continues to respond from a consensual impulse during near work by action of the sphincter iridis in contracting the pupil, thus, in a measure at least, lessening the useful effect of removing the supposed harmful influence of accommodation upon an irritated or inflammed chorioid and retina.

For a long period of time the surgical treatment of myopia has also sought interference with the extra-ocular muscles, especially the inferior oblique, the operation being based on the belief that the myopic condition has been produced by undue extra-ocular muscle-pressure. The efficacy of sclerotomy for the radical relief of progressive myopia is extremely doubtful.

In some cases of malignant myopia it may be worth while to endeavor to promote lymph-stream flow, and thus attempt to remove some of the disturbing débris of inflammatory action, by means of subconjunctival injections of aseptic solutions of such drugs as chloride of sodium and bichloride of mercury. To be of any value, however, they should be passed, as is so infrequently done, as deeply as practicable into the orbital tissues. Iridectomy also has been suggested.

From time to time radical procedures for the removal of corneal astigmatism have been proposed, but, as the result of failure to give any useful result, they have as often fallen into disrepute and have been abandoned. At the present time much experimental work, conducted on plans of higher scientific basis, is being conducted; but with what practical clinical value still remains to be seen.

*After-Treatment of Ametropia.*—After thorough and proper correction

of ametropia and heterophoria, more can be done by betterment of the general condition and hygiene than can be accomplished by any form of local orthopædic or operative treatment. If possible, the patients should be given bright and happy surroundings. They should be taken away from introspection, and should not allow their minds to dwell on supposed conditions which if not thought of would never make their presence known.

The ocular apparatus should have proper periods of rest, so as to allow any undue congestion that has been produced by forced motor and sensory action to subside. The lymph-stream and the vascular circulations should be allowed opportunity to rid the tissues of harmful waste. The working materials, as it were, should be given time for thorough replenishment, and care should be taken to see that the eyes are permitted temporarily to break away from all their physiologic associations with related organs. If these precautions be taken, it is certain, even in this most trying climate of ours, that, beyond the ordinary results which are produced by time, the visual apparatus will function properly, comfortably, and harmlessly.

The eyes are to be properly used, not abused. Care must be taken that objects are not brought too close to them, as too near a working point employs a greater accommodative and convergence impulse than is necessary to produce sufficiently large retinal images for distinct and easy vision. Kallmann's head-rest or Durr's horizontal reading support is of value in some such cases.

Bad habits are to be searched for and prohibited. All prolonged near work should be eschewed during convalescence from any wasting or weakening disease, or when the physical powers seem to be below par. The objects to be gazed at should be clear. Print should be legible, the form of type should be plain and distinct, and the color of the background upon which anything is looked at, no matter what it may be, should, if possible, be devoid of vivid or harmful contrast. If the paper of a book be too white and too glaring, a sheet of thin neutral-tinted paper or card-board placed beneath each page as read is often most grateful to the patient.

The eyes should be frequently rested by gazing at vacancy, thus tending to relieve any muscle-strain, just as in travelling a long distance on foot it will be of advantage to break the trip by a series of walks, rests, and short runs, thus bringing the man to his journey's end much less tired than he would have been if he had traversed the same road steadily and unremittingly at one gait.

All cases of corrected ametropia and heterophoria should be periodically and sufficiently often examined, in order to remove all the latent tendencies of heterophoric eye-strain that have not been brought to light and have not been corrected by the lenses and prisms previously ordered.

If these rules be observed according to the special indications in each individual case, there will remain but few—a very few—instances in which persistent and intelligent effort is not able to produce a well-working and a painless ocular apparatus.

# OCULAR LESIONS DEPENDENT UPON DISEASES IN THE CIRCULATORY SYSTEM.

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A SPECIAL consideration of the diseases of the eye resulting from pathological changes in the blood and circulatory system is of great importance from a clinical stand-point; partly because they endanger vision and partly because they are frequently of significance in judging of the general health of the patient. Those portions of the eye especially whose metabolism is active are dependent upon a proper supply of blood, and those structures upon which the most important functions devolve will suffer greatest when the blood and the vessels are diseased or the circulation is disturbed; the retina and its continuation, the optic nerve, being the tissues that are the most affected. The intimate relation existing between these structures and the brain has led to the assumption that, in those changes in the retina and optic nerve which disturb the circulation of the blood and nutrition, similar disease will exist in the brain.

Having within the eye-ground a richly developed blood-system, it was expected that with the ophthalmoscope much could be learned of the blood-vessel system of the rest of the body. Through a study of the eye-ground, attempts have been made to establish the existence of general arterial sclerosis, general impoverishment of the blood, etc. When the blood is rendered impure by effete material of the body and toxines, important changes may be observed in the tissues of the retina. Finally, it has been attempted to study the diseases of the heart and aorta by observations upon the condition of the retina in these affections.

Whether, however, all of the above-mentioned efforts to make the eye an index to the conditions of the blood and its circulation are well founded, is questionable, and shall subsequently be thoroughly discussed and criticised.

## GENERAL CONSIDERATION OF THE CIRCULATION AND NUTRITION OF THE EYE.

The eye obtains its entire blood-supply from branches of both carotids. The external carotid sends branches from the face towards the eye, while the internal carotid reaches it by the ophthalmic artery. The free anastomosis which exists between the internal and the external carotid is of the greatest importance for the proper supply of the eye; this organ even receiving blood from the vertebral artery through the circle of Willis and also from the opposite side of the base of the brain.

After ligation of the internal carotid and ophthalmic arteries in a corpse, Elschnig[1] injected a colored solution into the internal carotid and the external maxillary arteries, and saw that the arteries of both orbits became filled. Pilz[2] in 586 cases of ligation of the common carotid found only 14 instances in which there were visual disturbances in the eye of the same side. According to Elschnig, in only ten instances did these result from the ligation, and but once was the vision permanently impaired. Schmitt[3] has collected 26 cases of recently reported ligations of the carotid without occurrence of visual disturbance.

Schultén showed that the blood circulation in the eye is subject to similar changes and influences as in the rest of the body. In addition, the reduced elasticity of the sclera, which is associated with quickly increased pressure, resists every sudden and decided increase of the blood-pressure within the eye, and modifies the harmful reaction which this delicately constructed organ would suffer from a strong blood-current. On the other hand, in consequence of the continuous pressure upon the intra-ocular vessels, the eye is less protected against sudden anæmia than sudden hyperæmia, notwithstanding the numerous collateral vessels.

Marked dilatation of the blood-vessels, especially those of the chorioid, increases the intra-ocular tension, and thus renders difficult the entrance of the blood into the eye. The blood in the chorioid acts as a regulator for the maintenance of uniform fulness of the retinal vessels. The entrance and exit of blood to and from the retina is very limited in comparison to other parts of the eye, as only one artery and one vein are present for that purpose. Neither, after obstruction of the artery or the vein, can a rapid equalization be effected through collateral supply, since, except for the connection formed between the retinal and ciliary vessels by a few fine branches at the entrance of the nerve, the two systems are entirely separate. (Leber, *loco citato.*)

The chorio-capillaris may play an important *rôle* in the nourishment of the retina; this is shown in those animals in which the retina is almost

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[1] Archiv für Ophthalmologie, Band xxxix.

[2] Archiv für klinische Chirurgie, Band xi. 1868.

[3] Statistische Bemerkungen über Ligatur der Carotis Communis; Inaugural Dissertation, Würzburg, 1887.

without vessels (Sattler[1]). It is likewise proved by an anatomical study of the human retina (H. Müller[2]), in which it is seen that the layer of rods and cones is nearer to the chorio-capillaris than to the retinal vessels. When the central artery of the retina of man becomes clogged, the nervous elements of the inner layer (brain layer) atrophy, the sensory epithelium remaining intact. The chorio-capillaris is sufficient for the nourishment of the epithelial layer, but is unable to maintain its function, as cessation of the circulation of the central artery gives rise to immediate blindness.

Continued pressure of the finger on the eyeball is soon followed by loss of vision, this being due to the scanty supply of blood to the retina. The ophthalmoscope shows that the circulation of the blood in the central artery is intermittent at the disk, so that its termination is to an extent intermittently emptied, the blood having the power to enter only at the height of the pulse-wave.

To understand the disturbances of the circulation, especially in the retina, it must be remembered that the eye is not so richly supplied with blood as is often assumed. Axenfeld[3] called attention to the fact that only a very small part of the blood of the ophthalmic artery (about one-eighth) enters the eyeball, and that the eye *in toto* in comparison to its volume is poorly supplied with blood. The capillaries of the retina are the smallest in the body.

*It follows that hyperæmia or anæmia, occasioned by contraction or obstruction of the vessels, will be induced more readily and be more intense, the nearer to the eye the vascular disturbance occurs.*

The connection between the circulation of the eye and that of the brain, and their asserted interdependence, are not so close as was assumed by von Jäger, Bouchut, and others. On the other hand, a certain relationship of the circulatory process cannot be denied. Schultén (*loco citato*) has arrived at the conclusion that increased supply of blood to the brain (collateral circulation) cannot well occur without similar increase to the eye, and *vice versa.* The eye also shares in the anæmia resulting from weakened force of the heart's action or from diminished volume of blood. A spasm of the vessels of the brain, however, need not be accompanied by a like condition of the intra-ocular circulation, and similarly the cerebral vascular channels may be paralyzed without the appearance of the same condition of the vessels of the eye. Passive cerebral hyperæmia from interference with the venous circulation need not affect the eye, as the venous blood of this organ has other channels of emergence. Thrombosis of the cerebral sinuses may also exist without the circulation of the eye being seriously affected. It is probable that much that can be demonstrated upon animals is not discoverable with the ordinary clinical methods that are now available.

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[1] Archiv für Ophthalmologie, Band xxii, II., S. 88.
[2] Gesammelte Schriften, S. 137.
[3] Archiv für Ophthalmologie, Band xl. S. 111.

## DISTURBANCES OF THE CIRCULATION.

### ABNORMAL DILATATION OF THE BLOOD-VESSELS OF THE EYE.

#### HYPERÆMIA.

Pathological dilatation of the blood-vessels is usually the result of inflammation. Mechanical stasis of the venous blood through interference with its exit is of frequent occurrence. More rarely it appears as a result of plethora or from abnormal functional activity, paralysis of the vessels, etc.

Active hyperæmia from increased force of the circulation (hypertrophy of the heart, Graves's disease, etc.) is of infrequent occurrence. Passive hyperæmia may be acute or chronic in type.

If the obstruction be at some remote point, acute hyperæmia rarely injuriously affects the interior of the eye. It is exceptional for hemorrhagic extravasation to occur, when the blood-vessels are healthy, during whooping-cough, epileptic seizures (Gowers [1]), and strangulation, as in a case that was recently seen by the author. Neither Dyer [2] nor Green [3] found intra-ocular hemorrhages in subjects that had been executed by hanging, although the latter observer noted numerous minute hemorrhages in the conjunctiva. In strangulated rabbits and dogs Baquis [4] found serous infiltration of all the elements of the retina with a few extravasations from the distended veins. Förster,[5] however, mentions the finding of numerous retinal hemorrhages after death from suffocation.

The effect, however, is different when the cause of the congestion is within the eye itself, as in the case of thrombosis of the central retinal vein, which will be described later.

The congestion resulting from collapse and from paralysis of the vessels belongs to the condition known as acute passive hyperæmia. This is observed,—

1. In sudden diminution of intra-ocular tension. That form of diminished tension caused by operative incision into the eyeball has practical significance, as reported by Leber.[6] The writer's observations in this line have been, that this hyperæmia in normal eyes is only slight, and that it is principally confined to the veins; only leading to extravasation of blood when the vessels are diseased, and in glaucoma. Retinal hemorrhages often occur in glaucomatous eyes after iridectomy, these being due to abnormal rigidity of the vessels (senile sclerosis). Under similar conditions disastrous chorioidal hemorrhages occasionally occur during cataract operations.

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[1] Die Ophthalmoscopie in der inneren Medicin, 1893.
[2] Transactions of the American Ophthalmological Society, 1866, p. 13, and 1869, p. 27.
[3] Ibidem, 1875, p. 354.
[4] Annali di Ottalmologia, 1891, p 421.
[5] Handbuch der gesammten Augenheilkunde, Band iii. S. 63.
[6] Ibidem, ii. p. 350.

2. Hyperæmia from paralysis of the vessels may follow temporary anæmia. Schultén found that when the flow of blood to the eye was diminished, great congestion and increased tension were often to be observed upon removal of the ligature. Baquis[1] observed similar results in rabbits, and Marckworth[2] has seen them in dogs. These facts explain the cause of many striking instances of congestion or hemorrhage, in cases in which anæmia was to be expected,—*e.g.*, in certain cases of embolism or thrombosis of the central vessels of the retina.

Chronic passive hyperæmia is not observed as frequently as would be expected. The congestion of the region of the superior vena cava in heart affections and in emphysema must be quite marked before it can extend to the retinal vessels. This picture is likely to be developed most clearly in congenital affections of the heart. Knapp[3] reports a case of enormously distended veins of the retina without valvular disease, but with general dilatation and hypertrophy of the blood-vessel system of the body. The markedly tortuous veins seen in hypermetropic eyes may be mistaken for hyperæmia of the retinal veins, as described and pictured by Chodin,[4] Mackenzie,[5] and others.

## ABNORMAL CONTRACTION OF THE BLOOD-VESSELS OF THE EYE.

### ANÆMIA.

In local anæmia of the retina this condition may be confined to the arteries, while the veins may be hyperæmic. This is the case in glaucoma, in new formations, and in inflammatory products, this being caused by pressure upon the central artery and veins. The veins, having thinner walls, are more easily compressed. Intra-ocular tension may also produce local anæmia,—*i.e.*, ischæmia of the retina. The arteries become small and the papilla grows pallid, and only at the height of the systole is any blood driven into branches of the central artery. With the contraction of the visual field attacks of momentary blindness ensue, and usually, at the same time, syncope occurs from insufficient blood entering the sinuses of the brain, thus permitting a threatened syncope to be recognized. (Wordsworth,[6] Knies.[7])

Contraction of the retinal vessels following irritation of the cervical sympathetic has been observed by Leber[8] in rabbits, and by Schäler in

[1] Annali di Ottalmologia, 1891, p. 269.

[2] Archiv für Augenheilkunde, Band x. S. 269.

[3] Transactions of the American Ophthalmological Society, 1870, p. 120.

[4] St. Petersburger medicinische Zeitschrift, 1875.

[5] Transactions of the Ophthalmological Society of the United Kingdom, 1884, p. 152.

[6] Royal London Ophthalmic Hospital Reports, 1863, p. 8.

[7] Die Beziehungen des Sehorgans und seiner Erkrankungen zu den übrigen Krankheiten des Körpers und seiner Organe, 1893.

[8] Handbuch der gesammten Augenheilkunde, Band ii. S. 358.

cats. Opinions vary as to whether the retinal arteries are narrowed in attacks of migraine.[1] The observation of Siegrist[2] on fleeting scotoma in an eye in which the arteries were strikingly narrowed requires further study.

The cause of the ischæmia of the retina has been described by A. von Graefe,[3] Knapp,[4] and others. The author has reported a case of binocular ischæmia of the papilla and retina following bromethyl narcosis, which was associated with monocular blindness. The condition is similar to that occasioned by quinine toxæmia, which will be considered later.

On the other hand, a high degree of anæmia of the retina may be present without considerable reduction of the visual acuity, as shown by A. Graefe[5] to have occurred in cholera patients. The functioning of the retina is dependent more upon a continuous circulation, even though it is weak, than it is upon any degree of fulness of the vessels.

As the condition of the blood in the eye may be different from that found in other parts of the body, any attempt to diagnose general anæmia by the ocular findings may often lead to error. In general anæmia we frequently see hyperæmia of the conjunctiva. Anæmia of the eye itself is often readily diagnosed by the condition of the conjunctiva, with more difficulty by that of the retina, while, in many cases, it is impossible to perceive a corresponding change in the chorioid. In anæmia of the chorioid it is not essential that the eye-ground should be pallid, as the color of the fundus is principally dependent upon the pigment that is contained in the retinal epithelium. Attention must therefore be directed principally to the tint of the disk, and to the color and fulness of the retinal vessels. It is not always easy to judge of the latter condition, as the vessels may still possess their normal width while the volume of the blood is small, being flattened by intra-ocular pressure. In these cases they are more apt to be transparent, so that the underlying structures shine through. It must not be forgotten that it is usually only in cases of great diminution of the blood-pressure that anæmia of the optic nerve and retina can be diagnosticated with certainty. The appearances that are generally met with in general anæmia, chlorosis, and pernicious anæmia will be described under the section, "Diseases of the Blood."

The dangerous, but fortunately rare, disease of the eye consequent upon *acute anæmia after extensive or repeated hemorrhages* remains to be described. In the history of sixty thousand cases that have been seen by the writer at the eye-clinic in the University of Zürich and in private practice, not one unquestioned instance is recorded. Fries[6] has collected one hundred and

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[1] Die Migraine, Wien, 1894, S. 27.

[2] Mittheilungen aus Kliniken und medicinischen Instituten der Schweiz, 1894.

[3] Archiv für Ophthalmologie, Band viii.

[4] Archiv für Augenheilkunde, Band v. S. 203, and Transactions of the American Ophthalmological Society, 1880, p. 93.

[5] Archiv für Ophthalmologie, Band xii. Theil ii. S. 198.

[6] Klinische Monatsblätter für Augenheilkunde, 1876.

six cases which have occurred during the past two hundred and thirty-five years. In eighty-nine and a half per cent. the visual disturbance was bilateral, and in sixty one per cent. both eyes became blind. This affection results more especially (sixty per cent.) from gastric, intestinal, and uterine hemorrhage, while that from artificial abstraction of blood is but twenty-five per cent.; epistaxis, seven per cent.; and bleeding from wounds, five per cent.; while but one per cent. resulted from pulmonic hemorrhage. Why the loss of a similar quantity of blood should in one case produce disturbance of vision and in another not, why sometimes both eyes and at other times but one eye should be affected, is at present unexplainable. The sight of healthy individuals is not so readily destroyed by loss of blood as in patients suffering with disease of the stomach and the bowels. The character of the hemorrhage has no influence either upon the production of unilateral or of bilateral blindness or upon the degree of disturbance. Samelsohn[1] and Förster[2] have reported cases in which, after severe bleeding from the stomach and intestines, there was not any disturbance of vision.

In Fries's cases no improvement appeared in forty-seven per cent.; betterment took place in thirty per cent.; and complete recovery occurred in twenty per cent. The time at which improvement commenced varied greatly; although in the majority of cases, especially in intestinal hemorrhage, it was delayed for many days, weeks, or even months. According to the observations of von Horstmann,[3] von Kries,[4] and others, many cases are left with marked defect in the visual field. Landesberg,[5] in an examination of a case upon the day following a severe attack of epistaxis, found the vision in the right eye still normal, while that in the left equalled one-half. Both papillæ were swollen and cloudy, with indistinct margins. The retinæ were diffusely hazed, the arteries being only slightly contracted, and the veins being dilated and tortuous. The vision of the right eye became normal, while in the left a neuro-retinitis with subsequent atrophy of the disk developed. Hirschberg[6] reports a similar case of severe hemorrhage from the stomach, in which there was a more or less decided atrophy of the papilla. He says that this can occur as early as the eighth day. In cases of this character Horner has found that the intra-ocular tension was perceptibly diminished on both sides, while the slightest pressure upon the globe produced a strong arterial pulse, and an emptying of the veins.

Many attempts have been made to explain the peculiar symptoms of the disease. Still the views and theories whereby the condition of the blood in the sheath of the optic nerve or a retro-bulbar neuritis (von Graefe) has been assumed, or the propulsion of arachnoidal fluid into the inter-

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[1] Archiv für Ophthalmologie, Band xxi. und iii.
[2] Handbuch der gesammten Augenheilkunde, Band vii S. 73.
[3] Klinische Monatsblätter für Augenheilkunde, 1878, S. 147.
[4] Archiv für Ophthalmologie, Band xxv.
[5] Klinische Monatsblätter für Augenheilkunde, 1877, S. 147.
[6] Ibidem, 1877, p. 56.

vaginal space of the optic nerve by an existing cerebral œdema (Samelsohn) has been supposed, have all been forced into the background by the results of the anatomical researches of Ziegler, in which he was led to the conclusion that these changes are caused by local poverty of the blood. Ziegler, however, resorts to the supposition of local contraction of the vessels to explain the fact that visual disturbance does not always appear after a severe hemorrhage, he believing that a certain disposition, which has its origin in a sensitive vaso-motor system, is required for its production. The frequently found atrophy of the optic nerve which occurs in the affection was demonstrated in a case of Hirschberg's [1] in which it was proved to be due to a severe inflammatory condition. The sclerosis of the retinal arteries which Rählmann [2] found in his case may in the judgment of the writer be attributed to concurrent parenchymatous nephritis. Further anatomical study is required to decide whether inflammatory appearances are absent in all cases or whether a regeneration that is dependent upon ischæmia is being dealt with. Caution must be exercised in estimating the pathological significance of the ophthalmoscopic findings. The writer believes that for the final occurrence of this pathological change in the retina and the optic nerve an abnormal condition of the blood is necessary.

Every physician should know that hemorrhage from the stomach, bowels, or uterus threatens an incurable blindness. Besides energetic treatment directed towards the hemorrhage, the opportune employment of intra-venous infusions of normal salt solution, together with the maintenance of the horizontal position, is indicated. Should vision be impaired or blindness have occurred, but little can be done to secure a favorable result. In such cases hypodermatic injections of strychnia are perhaps most strongly indicated.

Similar to the above-described disturbance of vision is that resulting from quinine toxæmia. Light was first thrown on the origin of this form of blindness through the ophthalmoscopic investigations of Roosa,[3] Grüning,[4] Michel,[5] Knapp,[6] Voorhies,[7] Horner,[8] Buller,[9] and others, who have shown that the principal ophthalmoscopic changes consisted in marked pallor of the disk associated with smallness of the vessels. In the absence of all inflammatory symptoms (Voorhies) the optic nerve and retina were absolutely bloodless. The papilla was "chalk-white" in tint, and not a trace of a blood-vessel could be seen either on the disk or on the retina. Buller and Grüning saw haziness of the retina in the macular region, causing the fovea to appear as a red spot. The slightest pressure on the eyeball suf-

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[1] Centralblatt für praktische Augenheilkunde, 1882, S. 22.

[2] Fortschritte der Medicin, 1889, S. 928.

[3] Archiv für Augenheilkunde, 1881, S. 222.

[4] Ibidem, xi. p. 145.

[5] Ibidem, xi. p. 151.

[6] Ibidem, p. 156, and Klinische Monatsblätter für Augenheilkunde, 1881.

[7] Journal of the American Medical Association, 1879.

[8] Klinische Monatsblätter für Augenheilkunde, 1881.

[9] Transactions of the American Ophthalmological Society, 1881.

ficed to empty the scarcely visible blood-vessels. The usual history of this affection is that after the ingestion of a large dose of quinine or the use of rapidly repeated moderate doses of the drug, bilateral impairment of vision, together with tinnitus, vertigo, and mydriasis, appears; this usually being rapid, and in some cases passing on to complete blindness.

As a rule, the loss of vision caused by quinine is transitory. The greatly contracted field widens, the markedly disturbed color-perception returns, and after varying periods visual power generally attains to the normal. In the second stage the ophthalmoscopic appearances are those of optic atrophy with obliteration of the retinal vessels. Becker[1] and Brunner[2] have proved, by experiments made upon dogs that were blinded by quinine, that all the retinal vessels became bloodless and appeared as fine white streaks. Ischæmia, the same as that seen in man, appeared whenever the dose of quinine was repeated. Barabaschew[3] experimented upon six healthy human beings with doses of .3 to 2.4 to 3.6 grammes, and obtained symptoms that are analogous to those seen in cases of quinine amaurosis.

The result of the pathological conditions that have been studied in dogs by de Schweinitz[4] showed, as the result of blindness from long-continued doses of quinine, thrombosis of the principal vessels of the papilla. This was associated with an endovasculitis and an obliteration of the central artery of the retina, followed by atrophy of the optic nerves that extended to the tracts. He assumed that the drug affects the vaso-motor centre, producing constriction of the vessels with resulting changes in the vessels themselves, to which thrombosis may be added. Holden's more recent studies,[5] however, most properly place the earliest recognizable pathological signs in the ganglionic cells of the retina. Horner supposed that an endovasculitis resulted in consequence of the absence of blood from the vessels. Brunner considers it unlikely that the ischæmia of the retina and optic nerve results from spasm of the arteries, believing it to be due to lowered blood-pressure. According to investigations made by Briquet, Lewizky, and Schroff, a decided lowering of the general blood-pressure follows the administration of large doses of quinine, while the entrance of arterial blood into the eye upon which intra-ocular pressure is exerted is impeded, thus allowing ischæmia to follow.

The prognosis of quinine toxæmia is favorable. The object of the treatment should be to facilitate the flow of blood to the eye by rest in the horizontal position and by stimulants to the circulation. Digitalis is indicated to increase blood-pressure. Nitrite of amyl is of little value. Locally, repeated paracentesis of the anterior chamber should be recommended in every case.

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[1] Klinische Monatsblätter für Augenheilkunde, 1881, S. 104.
[2] Ueber Chininamaurose, Zürich, 1882.
[3] Archiv für Augenheilkunde, 1891, S. 91.
[4] Transactions of the American Ophthalmological Society, 1891.
[5] Ibidem, 1898.

Salicylate of sodium appears to be capable of affecting the eye in a similar manner, but such results have been observed more rarely than from quinine (Gatti).[1] Ziegler (*loco citato*, p. 71) believes that in many cases of lead-poisoning local anæmia plays an important part.

## CIRCULATORY PHENOMENA WITHIN THE EYE IN DISEASES OF THE HEART.

Diseases of the heart, especially valvular affections, frequently induce some alteration in the circulation of the blood within the eye, but, as a rule, it is difficult to see these changes distinctly, and for that reason they are of but slight value in general practice. An accurate knowledge of the normal condition, especially the pulse-appearances in the vessels of the fundus, is to be gained only by tedious investigation. Nothing in ophthalmology tires one so much as the careful study of the pulse-phenomena in the retinal vessels.

The proper position of the surgeon and the patient is of importance. If the patient lies in bed and the surgeon is compelled to lean over, the examination is rendered difficult, and only the most marked pulse-phenomena can be perceived. It is necessary that the patient, the examining instrument, and the observer's eye, be made absolutely steady, especially when the pulsation is weak. The gentle pulsatory movement of the upper part of the body of the patient or in the arm of the observer may imitate a very slight pulsation in the vessels in the observed eye. The upright image should be employed, as it affords the necessary magnification. Whenever it is possible, the patient should be seated with his arms upon a table. The observer should also have a support for the upper part of his body, or at least for his arms. Through the medium of "auto-suggestion" an observer is in danger of seeing pulsation when it is not present, especially if at the same time he feels the patient's pulse. It is better for the surgeon to count the observed pulse aloud, and allow an assistant to compare the count with the patient's pulse at the wrist. The patient must be capable of holding the eye absolutely still for a brief period of time. For this reason an accurate study sometimes fails when the vision of the other eye is poor or when the patient is not sufficiently intelligent to fix a certain object steadily.

The normal pulse-phenomena form one of the most attractive objects in ophthalmoscopy, especially as their explanation is somewhat difficult. A few words in reference to the various forms of these phenomena as met with in the retinal vessels are indispensable.

In the arteries and veins a form of pulse is to be observed which can be described as an arterial or venous end-pulse, being visible only at the termination of the vessels upon the papilla. A more appropriate term for this character of pulse is "intermittent inflow and outflow." This occurs usu-

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[1] Gatti, Gazzetta degli Ospitali, 1880, No. 4.

ally in the principal branches of the central artery when the intra-ocular tension is absolutely or relatively too high to the arterial pressure. Pressure of the finger upon a normal eye or the elevated tension of glaucoma induces this intermittent flow. When the blood-pressure sinks rapidly, as in commencing syncope, the normal intra-ocular tension becomes relatively too high. In all these cases the arterial blood is capable of entering the eye only at the height of the pulse-wave. A new growth behind the eyeball might produce this end-pulse, although the writer is unaware that such an observation has been made.

Arterial pulsation can generally be observed by the inverted image. Venous end pulse or intermittent outflow of the blood, first observed by van Trigt[1] and Coccius,[2] and afterwards by von Jäger,[3] von Graefe,[3] Donders,[4] and Helfreich,[5] is more difficult of explanation. This phenomenon, which occurs frequently in normal eyes, does not take place so quickly as is the case with the arterial end-pulse. Van Trigt has observed that the dilatation of the end of the vein occurred synchronously with that of the radial pulse. This to-and-fro movement of the blood-column can also be observed by the indirect method. Lang and Barrett,[6] in their observation of sixty-one normal eyes in persons from eleven to sixty-five years of age, having varying refractive conditions, found distinct venous pulse in seventy-three and eight-tenths per cent., doubtful in fourteen per cent., and absent in eleven and four-tenths per cent.

It requires a certain configuration of the disk end of the vein for the production of the pulse, it occurring particularly when a distinct infundibulum for the vessels, into which the venous branch descends, is present. Especially is this so when the cup has a somewhat abrupt bend, at which point the pulse may be particularly observed. Coccius gives the following explanation of the origin of this venous end-pulse. When through systole of the heart the arteries of the eye are dilated an increase in the intra-ocular tension exists, this pressure acting more forcibly on the parts that offer the least resistance, which in this case are the veins. In consequence the veins are constricted and the outward flow of the blood is quickened. Donders's (*loco citato*, page 94) explanation is somewhat different. This observer does not believe that the increased pressure affects all the veins alike, but supposes that the principal branches first suffer compression, and, in consequence, the collapse of the smaller branches is rendered impossible. In this way, he says, the circulation within the capillaries which nourish the tissues is not interrupted. Von Jäger[7] assumes that the diastolic dilatation of the

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[1] Nederlandische Lancet, 3d Series, 2d year.

[2] Ueber die Anwendung des Augenspiegels. Leipzig, 1853.

[3] Archiv für Ophthalmologie, Band i. S. 385.

[4] Ibidem, 1855, i. p. 75.

[5] Ibidem, xxviii. p. 8.

[6] Royal London Ophthalmic Hospital Reports, No. 12, p. 60.

[7] Wiener medizinische Wochenschrift, 1854.

central artery compresses the central vein within the sclerotic canal and dams back the venous blood. Jacobi,[1] on the contrary, considers the damming back of the venous blood to be due to a pulsatory recession of the papilla and of the physiological excavation. The theory of Helfreich[2] is based upon the investigations of Berthold (1869), Bergmann and Cramer (1873), which showed that systolic increase in pressure in the cerebral lymph is communicated to the veins which become compressed: in diastole of the heart they again become dilated. This rhythmical play between passive contraction and dilatation induces pulsation of the venous blood of the brain. As a result of the rhythmical increase in the flow of the blood in the cerebral arteries, the blood is, by compression, simply forced out of the cerebral veins and thereby produces a pulsatorily increased outflow of venous blood from the skull-cavity. Alterations in the blood-pressure of the cavernous sinus must influence the blood-circulation in the veins of the orbit and interior of the eye. The calibre of the vessels is reduced from the lowering of pressure occurring within the cavernous sinus from which an outward aspiration of the blood from the sinus results from existing anatomical conditions. Collapse can only take place in those portions of the veins which lie free upon the disk and whose walls are not bound down to the surrounding tissues.

Donders believes that the cause of the so-called venous end-pulse resides entirely within the eye, while Helfreich states that it lies external to the eye. The former assumes that the increase in tension on the entrance of the pulse-wave produces a momentary compression of the venous terminals, so that the outflow of the venous blood is somewhat arrested. Helfreich bases his view on the fact that during systole the pressure within the cavernous sinus is considerably increased, while during diastole it is markedly diminished. He supposes that the fluctuation in the pressure is communicated to the inosculating veins. During diastole, the flow of blood towards the cavernous sinus being increased, the central vein collapses. During systole the blood in the central vein must be dammed back; while during diastole the blood-pressure within the sinus sinks, and an increased outflow of blood from the portions of the veins that are coursing free upon the papilla takes place. On the other hand, an increased flow of blood towards these venous terminals cannot occur, as the retinal capillaries are very narrow. Consequently, at the moment that more blood flows from the central vein at the cerebral end than enters it from the retinal side, the papillary terminals are emptied to a considerable degree, and remain in a collapsed state until, as a result of the pulsatory dilatation of the cerebral arteries, the pressure within the sinus is again increased and the inflow of blood is retarded.

Before examining the explanations of the physiological pulse-phenomena, the observations of Wadsworth and Putnam (*loco citato*) must be cited.

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[1] Archiv für Ophthalmologie, Band xxii.

[2] Loco citato.

These authors saw rhythmical alterations of the venous pulse upon the disk of healthy men, the pulse becoming stronger or weaker in periods corresponding to about five respiratory movements; these results recalling "the rhythmical changes seen in connection with arterial tension that have been described by Traube, Hering, Cyon, and Sigmund Meyer, and which are most likely the cause of the long wave-movements of the brain spoken of by Mosso, Putnan, and others. These venous branches seem to pulsate under the influence of two wave systems,—the one which is synchronous with the heart-beat, and the other probably with the variation of the arterial tension." Notwithstanding the explanations of Debrowski[1] and van Trigt, the writer fully confirms these observations. This periodic pulse must originate from causes that are independent of the eye, and must result either through the rhythmical changes in the blood-pressure described by Traube-Hering or from an unknown cause.

According to the observation of the writer, the periodicity of the venous pulse has no connection with the respirations, they being somewhat irregular. In many cases, as above stated, they correspond to about five respirations, but variations from this occur. In reference to this question the writer would point out the possibility of the periods not being due to rhythmical changes in the blood-pressure as spoken of by Traube and Hering, but dependent upon rhythmical movements of the arteries, as have been observed in the ears of rabbits by Schiff. For the present, the writer will support the view held by Helfreich, and will attribute a secondary importance to the factors that have been considered by Donders. The two theories more or less substantiate one another, while perhaps individual differences play an important *rôle*. With his manometer, Schultén has been able to observe a decided change in the intra-ocular tension which was synchronous with the heart-beat, thus in a measure confirming Donders's theory.

In the judgment of the writer, the course of events in the production of an intermittent outflow of blood is as follows: when the intra-vascular pressure within the skull-cavity is increased by the cardiac systole, then the blood flows pulsatorily more strongly through the jugular veins (Berthold and Cramer). At this moment the blood in the retinal veins is dammed back while the pressure in the cavernous sinus is increased. According to Helfreich, the ends of the central veins must dilate in consequence of the damming back of the blood from the sinus. In accordance with Donders, constriction of the ends of the veins upon the papilla may result as a consequence of the systolic action, the off-flow of the venous blood being hindered in this manner for a brief period of time.

Actual pulsation of the retinal arteries and veins may be distinguished with certainty from the intermittent inflow and outflow, in the facts that they express themselves (1) by rhythmical alteration of the calibre of the

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[1] Münchener medicinische Centralblätter, 1870, S. 1.

affected vessels; (2) by movement, whether it be in that the arch of the vessels moves pulsatorily or that it produces a forward and a backward motion in the branch of the vessel. The rhythmical alterations of the calibre of the vessels are seldom visible under normal conditions, but are frequently found in heart affections. On the contrary, the movements may be often observed in the arteries of the normal individual, especially when the vessel describes a strong arc. The writer believes that in healthy subjects the pulse can nearly always be seen more or less marked in the arches of the larger retinal arteries. In certain heart affections it is plainly evident. The pulse-motion will not be visible in persons in whom the retinal arteries take a straighter course, even when favorable conditions are present. Strong heart action naturally favors the production of rhythmical alteration and movements in the vessels, whether in health or disease. In addition, the more firmly the vessels are bound to the tissues the less easy is it to produce a pulse alteration. Czermak[1] and Schmall[2] have observed marked rhythmical alteration in an arterial loop that projected into the vitreous humor, the other retinal arteries being pulseless. That these pulse phenomena are also related to the elasticity of the vessel walls has been established principally by Thoma.[3] Rählmann and his pupils have referred these pulse phenomena to the consistency of the blood.

Usually the pulse-wave ceases before it reaches such narrow arteries as the central artery of the retina, and intra-ocular tension also operates against rhythmical pulsation. If, however, the ordinary pulse-wave is abnormally great, then the central artery and its branches will show distinct pulsation, and also under some circumstances one corresponding with the veins and their branches.

In certain heart affections, such as aortic insufficiency, in which pulsation occurs even in the finger-nails, rhythmical reddening and bleaching of the disk, as has been described by Quincke,[4] Becker,[5] and Fitzgerald,[6] may be perceived. Here the pulsation of the retinal artery can be observed for some distance from the papilla, even to the branches of second degree. It is necessary for the production of these phenomena, however, that the pulse-wave in the aorta shall be abnormally strong and subside with unusual quickness. At times, in spite of a marked insufficiency of the aortic valves and decided dilatation and hypertrophy of the left ventricle, no retinal pulse (or slight at the most) can be seen. In other cases, however, with moderate degrees of valvular disease and with but little change in the heart, marked pulse-phenomena become perceptible. This result has led Thoma[7] to connect these cases with the arterio-sclerosis which supervenes

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[1] Centralblatt für praktische Augenheilkunde, 1883, S. 289.

[2] Archiv für Ophthalmologie, Band xxxiv.

[3] Ibidem, p. 85.

[4] Berliner klinische Wochenschrift, 1868, Nr. 64, and 1870, Nr. 21.

[5] Archiv für Ophthalmologie, Band xviii., I., 1872.

[6] British Medical Journal, December, 1871.

[7] Archiv für Ophthalmologie, Band xxxv.

upon valvular disease. He has produced artificial aortic insufficiency in dogs by perforating their aortic valves. On the following day tortuosity and pulsation of the retinal artery were developed, while the elasticity of the arterial walls became decidedly diminished. Subsequently distinct arterial sclerosis of the aorta could be demonstrated.

In well-marked cases of aortic insufficiency pulsation of the veins is sometimes found to be more pronounced than it is in the arteries. In regard to this, the writer can confirm the statements of Helfreich, Becker, Gowers,[1] and van Osten-Sacken. The last-named author explains these progressive peripheral pulsations in aortic insufficiency by stating that, during the heart's systole, the pulse waves enter the veins through the capillaries and there produce dilatation. This is followed by a contraction of the vessels during the heart's diastole, which becomes marked upon account of the pre-existing pathological enlargement of the capillaries.

In a case of aortic insufficiency, the writer has satisfied himself that the arterial and the venous pulse were synchronous while a simultaneous well-developed intermittent outflow existed in the capillary venous terminals. The intermittent diminution of the venous end-flow closely followed the pulsatory contraction of the vein. From the investigations of these observers already quoted, as well as those of Helfreich,[2] Schmall,[3] van Osten-Sacken,[4] and the writer, the other varieties of valvular heart-disease, as a rule, do not exhibit any visible pulsations in the retina. Still, Schmall (*loco citato*) reports one instance of arterial pulsation that occurred in mitral insufficiency, and the writer has seen one case appearing with mitral stenosis. In uncomplicated cases of heart-disease marked dilatation of the veins seldom takes place.

## DISTURBANCES OF THE EYE IN DISEASES OF THE AORTA, THE CAROTID ARTERIES, AND THE OPHTHALMIC ARTERY.

Embolism of the central artery of the retina resulting from disease of the heart and of the aorta and the carotid arteries will be considered later. As regards aneurism of the aorta, it becomes necessary still to wish, with Becker, that there were a much larger group of well-observed cases. In the autopsy of a case of aneurism of the ascending branch of the aorta, observed by Becker, not a trace of spontaneous arterial or venous pulse was visible. According to this authority, the possibility of determining the location of an aneurism of the aorta by the circulatory phenomena present in the two eyes must be considered. Aneurism may produce a regurgitation of the blood from the carotid artery, giving rise to a pulsation of the retinal arteries, but, from the nature of the affection, it follows that

[1] Die Ophthalmologie. etc., S. 23.
[2] Festschrift zur Frederic Horner, 1881.
[3] Archiv für Ophthalmologie, Band xxxiv. S. 1.
[4] Loco citato.

the appearance of the retinal vessels may be different,—namely, when at the same time aortic insufficiency exists along with the aneurism.

In two cases the following observation was made by the writer. The first patient, with an aneurism of the ascending aorta, showed normally filled retinal arteries and veins without pulsation in the right eye. Intermittent entrance of the blood only occurred when ordinary pressure was made. In the left eye the veins were distinctly enlarged, only a slight pressure on the globe being required to produce an intermittent flow of blood in the arteries. If a pressure that was sufficient to induce an intermittent arterial pulsation in the normal eye was exerted on the left one, the arterial terminals became empty. In this case the blood-pressure in the left carotid and the left retinal arteries was manifestly diminished, and, in consequence, the retinal arteries were dilated. In the other case of aneurism of the aortic arch, the left pupil was contracted from paralysis of the sympathetic nerve, but the palpebral fissure was not narrowed. No pulse could be determined in either the carotid or the brachial arteries, but it could be felt in the crural arteries. This patient was a syphilitic and had bilateral pigmentary chorio-retinitis of the entire fundus. The disks were normal. With pressure on the ball, the blood-column at the venous ends became granular, and a similar reflex, which was already narrow and faint, became narrower and less distinct. At the papillary end of the venous branches the granular contents of the veins could be seen to flow centripetally. By increasing the pressure the contents of the vessels became brighter and more granular and the velocity of the current decreased. If the pressure was removed the current rapidly quickened. The section of the vessels in which the current was present became darker red and the visible flow ceased. In this case a markedly decreased blood-pressure in the carotids and retinal arteries, by which an artificially increased intra-ocular tension alone stopped the circulation of the blood in the retinal vessels, was that which was being dealt with. It is remarkable that this scanty blood-supply produced no permanent disturbance, notwithstanding the fact that the slightest hinderance to the circulation was sufficient to induce momentary blindness.

The other ocular symptoms arising from aneurism of the aorta result from paralysis of the sympathetic nerve, and manifest themselves in vaso-motor disturbances, including contraction of the pupil and palpebral fissure, upon the affected side. Ogle[1] has published fifteen cases of this nature.

Paralysis of the sympathetic nerve is rarely caused by an aneurism of the common carotid[2] or of the internal carotid arteries.[3] Frequently these affections are unaccompanied by visible changes in the eye of the same side. The author has observed in a case of aneurism of the external carotid artery an absence of venous end-pulse in the corresponding eye, which

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[1] Transactions of the Medico-Chirurgical Society, 1858.
[2] Ibidem. [3] Ibidem.

phenomenon was very evident in the opposite organ. When aneurismal dilatation of the carotids is present on both sides, the ocular disturbances prove, as has been observed by Michel,[1] to be more striking. Marked deterioration of vision and decided fulness of the veins, with scattered areas of opacity in the retina, were observed. Michel has referred to the choked disks which were present in the changes found in the carotid arteries, these conditions interrupting the flow of lymph from the lymph-spaces of the optic nerve towards the brain. The so-called arterio-venous aneurism between the internal carotid artery and the cavernous sinus which produces pulsating exophthalmus, may occur spontaneously or can be produced by traumatism, the essential lesion being a rupture of the carotid artery into the cavernous sinus. (See article on Diseases of the Orbit.)

Owing to its extreme rarity, true aneurism of the ophthalmic artery plays an unimportant part. It can produce the same symptoms as the previously described affection. Strong compression of the ophthalmic veins may produce marked stasis in the veins of the orbit (Guthrie).[2] Pseudo-aneurism of the ophthalmic artery and diffuse pseudo-aneurism of the orbit must be extremely rare (Sattler, *loco citato*). The same is true of arterio-venous aneurism of the orbit and of cirsoid or racemose aneurism. Varicose dilatation of the ophthalmic vein is also an unusual affection. Panas[3] cites but thirteen instances to be found in medical literature.

Only a few cases of thrombosis of the carotid and thrombosis and embolism of the ophthalmic artery have been described. Steffan's[4] assumption that the cases of embolism of the central artery are instances of embolism of the ophthalmic artery has not been supported by recent investigations. Fränkel,[5] Uhthoff,[6] and Elschnig[7] have proved that obstruction of the ophthalmic artery from thrombosis has but little injurious effect upon intra-ocular circulation, although it may produce marked temporary visual disturbance. Elschnig (*loco citato*) also shows the same to be true of thrombus of the internal carotid artery or even the common carotid artery. The observations of Michel[8] in a case in which the ophthalmoscopic appearances were those of embolism of the central artery of the retina, but the lesion was that of thrombosis of the internal carotid, prove nothing adverse to what has just been said, as the author himself admits the possibility of a piece of the thrombus having become detached and lodged in the central artery, in which position it became subsequently absorbed. In

[1] Festschrift zur Frederic Horner, 1881.

[2] Lectures on Operative Surgery of the Eye, London, 1873; Handbuch des gesammten Augenheilkunde, Band vi. S. 864.

[3] Traité des Maladies des Yeux, Paris, 1894.

[4] Archiv für Ophthalmologie, Band xii.

[5] Archiv für pathologische Anatomie und Physiologie, und für klinische Medicin, Band lxxix.

[6] Archiv für Ophthalmologie, Band iii., I., S. 23.

[7] Ibidem, xxxix. p. 84.

[8] Festschrift zur Frederic Horner.

Gowers's[1] case of partial thrombosis of the internal carotid artery and the ophthalmic artery, in which the eyeball was destroyed, a septic embolus may have produced the destruction. The results following ligation of the common carotid artery speak against an obstruction of these vessels producing serious injury to the eye. Likewise, compression of the common carotid artery in the neck, which Elschnig practised upon himself, did not produce any change in the retinal circulation or in the functioning of the retina itself.

Thrombosis of the cavernous sinus will not be considered in this connection. In rare instances, spontaneous thrombosis is induced in this locality by marasmus arising from exhausting diseases, as has been observed by Heubner,[2] Huguenin,[3] and Coupland.[4] Thrombosis from injury is just as infrequent. The thrombotic process may extend from the orbital veins into the sinus, or *vice versâ*. Simple thrombosis of a cavernous sinus does not produce congestion of the veins of the orbit, and, consequently, exophthalmus only results when the inferior and the superior ophthalmic veins are also thrombosed.

## CIRCULATORY DISTURBANCES IN THE AREA SUPPLIED BY THE CENTRAL ARTERY OF THE RETINA FROM EMBOLISM AND THROMBOSIS.

In embolism and thrombosis of the central artery there is found the most pronounced anæmia of the retina, with the exception of the variety which follows the rare lesion of laceration of the central artery from forcible separation of the optic nerve of the globe. The complete blindness which follows rapidly upon total obstruction of the central artery reveals the importance of the supply of the blood to the retina. This function is never regained, even though the obstruction lasts but a brief time and is soon followed by restoration of the circulation. It is only in a few cases that very slight vision is retained or is regained during the course of the disease. Many points, however, in reference to these affections have not yet received satisfactory explanation. The question in which cases the symptoms of obstruction of the central artery are to be ascribed to embolism and which to thrombosis still requires decisive answer. In a majority of instances the explanation favors the occurrence of embolism. In A. von Graefe's,[5] in Schweigger's,[6] and in Sichel's[7] cases, an embolus in the lamina cribrosa was demonstrated to be the cause of the obstruction. Priestley Smith[8] found, four months after the appearance of an embolism,

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1 Die Ophthalmoscopie, S. 87 und 169.
2 Archiv der Heilkunde.
3 Pathologische Beiträge, Zürich, 1869.
4 The Medical Times and Gazette, 1881.
5 Archiv für Ophthalmologie, Band v. S. 1.
6 Ueber dem Gebrauch des Augenspiegels, Berlin, 1864.
7 Archives de physiologie normale et pathologique, 1871.
8 British Medical Journal, 1874, p. 452.

that the artery was obstructed immediately behind the eyeball. Schmidt-Rimpler[1] has demonstrated the presence of an embolic-thrombotic closure of the central artery immediately after its entrance into the optic nerve. In a microscopic examination made by Nettleship[2] some five or six months after the occurrence of blindness, he came to the conclusion that a thrombosis had been added to an embolism, glaucoma being present. On the other hand, in a case reported by Loring,[3] Delafield found, eleven months after an embolism had been diagnosed, that the central artery and vein were nearly empty. The cases of Popp[4] and Hirschberg (Becker)[5] have shown that three years and five months respectively after an embolism, the vessels were normal and their lumen was free. Schnabel and Sachs[6] found an embolus in the portion of the central artery lying between the layers of the lamina, two-thirds filling the lumen, with a second embolus in a branch of the inferior artery at the point where it crossed the papilla. One year after the primary affection Manz[7] found in a glaucomatous eye an embolism that failed to present any definite structure. The mass was lying free in the artery in the anterior portion of the lamina cribrosa. Eight weeks after the occurrence of an embolism, Gowers[8] saw an oblong granular embolus situated behind the lamina cribrosa, with small fragments in the contracted arteries on the papilla. Similar cases have been reported by Wagenmann,[9] Uhthoff,[10] and Elschnig.[11] Marple[12] has seen in a longitudinal section of an eye that was removed for glaucoma (seven weeks after the obstruction of the artery) an embolus situated immediately behind the lamina cribrosa; posteriorly in the central artery there was a thrombus. In these sixteen cases in which anatomical studies have been made, the obstruction of the central artery occurred, in most of the cases, in the vicinity of the lamina cribrosa. In three instances only (Loring, Popp, and Hirschberg) no embolism could be discovered. In one case reported by Nettleship, and one noted by Schmidt-Rimpler, primary thrombosis was suspected. The anatomical material at hand has been insufficient to permit of a conclusion as to what part primary thrombosis plays in the occlusion of the artery; the want of proper answer being further emphasized because the microscopic examinations were not made for a long time after the occurrence of the

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[1] Archiv für Ophthalmologie, 1874, Band xx.

[2] Royal London Ophthalmic Hospital Reports, 1874, p. 9, 1875, p. 251.

[3] The American Journal of the Medical Sciences, 1874, April.

[4] Dissertation, Regenberg, 1875.

[5] Centralblatt für praktische Augenheilkunde, 1884, Band viii.

[6] Archiv für Augenheilkunde, 1885.

[7] Festschrift zur Feier des siebzigsten Geburtstages von Hermann von Helmholtz, 1891.

[8] The Lancet, 1875, and Die Ophthalmoscopie, S. 41.

[9] Archiv für Ophthalmologie, Band lx.

[10] Archiv für Augenheilkunde, 1894, S. 62.

[11] Ibidem, 1892, p. 4.

[12] New York Eye and Ear Infirmary Reports, 1895.

blindness, thus permitting post-mortem changes that might invalidate results.[1]

Nevertheless, these few anatomical studies have led to the belief that the ophthalmoscopic appearances known as embolism of the central artery of the retina actually result either from an embolism or a thrombosis, thus greatly weakening any hypothesis that they are due to embolism of the ophthalmic artery (Steffan)[2] or to hemorrhage into the sheath of the optic nerve (Magnus).

While the embolic nature of the process has not been absolutely disproved, still the number of observations in which no possible source of origin for emboli could be found was large. Therefore the assumption is permissible that in these cases the obstruction was due to thrombosis, as has been stated by A. von Graefe. Inflammation in the vicinity of the central retinal artery need not, however, produce thrombosis in the vessel, as in the inflammatory processes in the head of the optic nerve symptoms of obstruction of the artery are, as a rule, wanting. It is necessary, however, to take into consideration the local conditions that are present in which the central artery entering the eyeball divides into branches at this point. The vessel is also subjected to intra-ocular tension, and, consequently, it is reasonable to believe that these two factors, together with the motion which the optic nerve at its ocular insertion continually undergoes during the movement of the globe, can give rise to certain alterations in the vessel walls which, under some conditions, may produce a thrombosis. It must also be assumed, as will be seen later, that such local predisposing conditions in the central retinal vein in which thrombosis may appear play an important *rôle.*

Among other authors who express a doubt as to the obstruction of the central artery always being due to embolism are Mauthner,[3] Loring,[4] Nettleship,[5] H. D. Noyes,[6] Wadsworth,[7] and Priestley Smith.[8] The last-named author asserts that medical aid is of more service in thrombosis than it is in embolism. He also states that in cases in which thrombosis has declared its presence by transient obscuration of sight, he has been able, under certain circumstances, to avert blindness by increasing the heart's action. From a diagnostic stand-point he considers the obscuration of vision, which in many

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[1] It appears to the writer that the investigations are not always properly performed. His experience has been that it is better to make the sections at right angles to the vessels, and not longitudinally, as has been done in most instances. Longitudinal sections do not afford sufficient information as to the contents of the vessels, while in transverse ones the topography of the changes can be more readily studied. Usually, the most important findings are those that are obtained by a combined study of the conditions that are contained in two or three sections.

[2] Archiv für Ophthalmologie, Band xii , I.

[3] Medicinische Jahrbücher, Wien, 1873, S. 195.

[4] American Journal of the Medical Sciences, vol. lxvii.

[5] British Medical Journal, 1879.

[6] Transactions of the American Ophthalmological Society, 1888, p. 98.

[7] Ibidem, 1890, p. 672.

[8] The Ophthalmic Review, 1884, vol. iii. p. 1.

cases precedes the obstruction of the artery, of most decided significance. He asserts that this temporary darkening may occur much earlier in beginning thrombosis than it does in embolism. Similar cases have been seen by Parinaud[1] and Galezowski.[2]

The writer's views on this subject have found expression in a measure in the work of Kern,[3] who has collected the cases of embolism from the eye-clinic in the University of Zürich between the years 1864 and 1891, and in the writer's private case-books from the year 1878 to the year 1891. Out of a total of fifty-four thousand eight hundred eye cases, only twelve instances of so-called embolism of the central artery of the retina were found, and in but two of these cases was an undoubted source of the origin of embolus obtained. Thus, in eighty-three per cent. of these twelve cases no positive cause for embolus could be detected. The same author has collected eighty-three cases of so-called embolism of the central artery from medical literature, in which the heart and vascular conditions were noted, and as a result he has found that in sixty-six per cent. no positive causative factor for the origin of the embolism could be determined. On the contrary, a diseased condition of the peripheral vessels which might give rise to a thrombus seemed to exist in some of the cases. In sixty-three instances in which the author could not find any positive cause for embolus, there were twenty-four examples of sclerosis of the peripheral arteries, five of chronic nephritis, and six of syphilis. These facts have led Kern and the writer to the following conclusions: 1. That a majority of the cases that present the picture of embolism of the central artery are not due to an embolus, but are probably dependent upon a local disease of the central artery, such as atheroma, syphilis, endarteritis from chronic kidney-disease, or other dyscrasia. 2. That syphilis should be considered more commonly the cause of this affection, especially in cases in which there is not any anomalous heart affection.

Since examining anew all the literature pertaining to this affection, *the writer not only reiterates* his expressed opinion, but goes a step further, and says *that the entire theory of embolism of the central artery of the retina requires fundamental verification in order that light may be shed upon many of its obscure points.* In the writer's opinion, not once in the above-cited fourteen anatomical studies has sufficient proof been presented that in the majority of cases embolism was present; moreover, in a large proportion of the cases previous endarteritis and thrombosis had been active in producing obstruction of the vessel. All reports based upon a study of longitudinal sections must be accepted with caution. The theory of partial embolism of the central artery (a very questionable condition, and one that is difficult to understand) has also been advanced to explain certain symptoms of so-called embolism. It is necessary, however, here, to assume a

[1] Gazette médicale de Paris, 1882, No. 50.
[2] Traité d'Ophthalmoscopie, 1886, p. 165.
[3] Zur Embolie der Arterien Retinæ. Inaugural Dissertation, Zürich, 1892.

partial obstruction in a majority of these cases in order to harmonize the symptoms of an embolic process, but neither this theory nor any other concerning embolism is sufficient to explain the precursory changes and the symptoms of the various cases.

In general, only non-infected emboli are here considered, this subject-matter treating only of such emboli as are met with in heart-disease, in aortic aneurism, or perhaps in atheroma of the larger arteries. Further, a thrombotic clot may act as an embolus. The possibility of a so-called "crossed" embolus[1] must be considered,—that is, in case of an open foramen ovale of the heart, a thrombus originating in a vein may be carried into the carotid or its branches. Further, a parenchymatous cell-embolus[2] may result; a hepatic, a placental, or a bone-marrow cell may be borne away and form an embolism, and eventually also a "crossed" embolism may take place. Finally, a fat embolus may be observed in the eyes, although none as yet has been described. Besides the known causes of fat emboli, laceration or inflammation of the bone-marrow, inflammation of the subcutaneous and pelvic fatty tissue, contusion or necrosis of the liver, and simple severe contusion of the bones (according to Ribbert)[3] may produce the condition.

It is necessary to differentiate between emboli of the trunk of the central artery and those of the branches, since different results, both anatomical and clinical, follow the two forms.

### I.—OBSTRUCTION OF THE TRUNK OF THE CENTRAL ARTERY.

In this condition, quite suddenly, complete or almost complete blindness occurs in one eye. This condition may be observed on awakening in the morning, or noticed following severe physical exertion. If the blindness does not occur during sleep, the attack is generally accompanied by marked light and color phenomena. Speedily, however, all objects appear enveloped in a gray or a tinted mist. In some cases the obscuration of the visual field will proceed from the periphery towards the centre, while in others it will extend from the centre towards the periphery. In many patients *permanent blindness is preceded for several weeks or months by a transient disturbance of vision in the affected eye, this appearing in the form of a complete or a partial blindness which lasts for but a few minutes or hours at a time.* There can be no doubt that such prodromal obscurations of sight occur also in the other eye, either simultaneously[4] or while a similar condition still persists in the primarily involved eye. It may also appear in the second eye, after the eye that has been primarily attacked has recovered. This combined disturbance calls for special consideration, which will be given later.

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[1] Berliner klinische Wochenschrift, 1894, Nr. 1.

[2] Fortschritte der Medicin, 1893, Nr. 20.

[3] Correspondenzblatt für schweizer Aerzte, 1894.

[4] American Journal of the Medical Sciences, vol. lxvii., 1894.

At this period a marked anæmia of the retinal vessels may be noticed. The vessels form narrow red lines whose ramifications become increasingly finer until they disappear. In many cases the arteries in the vicinity of the papilla are demonstrable only as bright white streaks. The veins usually show a larger calibre, but are narrower than normal; especially is this so upon the optic disk. Towards the periphery, on the contrary, their blood-diameter is greater. Frequently, upon and in the vicinity of the disk, the arteries and the veins cannot be differentiated, the light-streaks being wanting. There is an absence of pulse-phenomena which is quite significant. An intermittent flow of blood in the arteries cannot be induced by pressure upon the eyeball. The flow of arterial blood to the eye is either abolished or it is greatly diminished.

Narrowing of the branches of the central artery is most distinctly seen upon the papilla, which, as a rule, is pallid during the early stages of the affection. In some cases, in which there is a complete annihilation of the retinal circulation, normal tint of the disk may be observed. An important symptom of obstruction of the central artery is the usually rapid onset of an opalescence of the retina that involves only those portions of the retina that possess the greatest thickness,—that is, in the vicinity of the macula lutea and the zones that surround the papilla. For this reason, the middle of the fovea centralis presents a deep-red, ill-defined spot which was formerly considered as a hemorrhage. In reality it is the chorioid that shows through the opalescent retina, allowing its color to be intensified by contrast. Should the chorioid be highly pigmented, the spot will appear dark, as Inouye[1] has observed in a coal-black Japanese. The same appearance would present itself in Negroes. The densest haze is situated around the margins of the fovea, and in this region the retina appears of a milk-white tint.

By feeble illumination the opacity assumes a bluish or a greenish tint, and by intense illumination it appears a dense white with bright stipplings and brilliant points. From the macula in all directions, except towards the papilla, the opacity fades. The circumpapillary opacity frequently diffuses itself in varying degrees as fine radiating striations. Towards the periphery the hazy retinal area gradually lessens until the normal red reflex of the eye-ground can be recognized. When the opacity does not veil the vessels it frequently causes them to assume a peculiar appearance, so that by some the arteries are stated to be as dark as the veins usually are. In the macular region the finer vessels are unusually dark and prominent. The opacity of the retina may be distributed irregularly in accordance with the restriction of the flow of blood of the retina. The retina itself may be somewhat thickened in the portions that show the greatest opacity.

The haze may appear very quickly after the obstruction of the artery.

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[1] Centralblatt für praktische Augenheilkunde, 1892, S. 320.

Schnabel and Fischer[1] have seen it after two hours' time, and Mittendorf[2] has noticed it after three hours' time from the onset of the blindness. Rarely does the opacity appear later than the second day after the onset of the blindness. In the first case of embolism observed by von Graefe (*loco citato*) the opacity of the retina did not come on until the eighth day, and he records eighty cases in which the opacity appeared within the first week. (Fischer, *loco citato*, p. 126.)

The changes that are observed during the course of the affection, especially those that are situated in the vessel-system of the retina, are of particular interest. It is frequently noticed that the arteries soon begin to refill. Schnabel reports a case in which, fifteen hours after an attack of blindness, the supply of blood to the retina was suspended. Nine hours later, however, a feeble circulation, with an appearance of the vessels reassuming their normal calibre, established itself. Von Graefe and Zehender[3] also report instances of this type.

While in embolism of the central artery of the retina late marked arterial and venous anæmia might be expected, the fact is that this has occurred to a greater or less degree immediately after the occlusion. Absolute anæmia of the central artery and its branches is very rare. (Fig. 1.)

The return of blood to the retina is shown in many cases by a peculiar breaking up of the blood-column in both the arteries and the veins, or a spontaneous pulse may soon appear. In most instances the venous pulse has shown itself in from four days to one week's time. After the refilling of the vessels, which may be very irregular, has somewhat improved, the intermittent flow of blood into the arteries can readily be induced by the slightest pressure upon the globe. The intermittent blood-column was first described by Jäger. It shows itself in the arteries, and frequently in the veins, as a breaking of the blood-column either into dark and light cylinders or into granules. As a rule, these cylinders or granules move more or less rapidly and ordinarily in the normal direction, but sometimes they may traverse a reverse direction. This latter condition has been observed by Hirschberg,[4] Elschnig,[5] Perles,[6] and Bjerrum.[7] At times these movements are seen in but one branch of an artery or vein, while occasionally they may take place simultaneously in both (Meyerhöfer,[8] Schell,[9] and Harlan[10]). A pulsatory advance of the blood-cylinders is but seldom seen (Morton).[11]

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[1] Ueber die Embolie der Arteria Centralis Retinæ, Leipzig, 1891.
[2] Transactions of the American Ophthalmological Society, 1892.
[3] Klinische Monatsblätter für Augenheilkunde, 1874, S. 210.
[4] Centralblatt für praktische Augenheilkunde, 1884, S. 71.
[5] Archiv für Augenheilkunde, Band xxiv. S. 67.
[6] Centralblatt für praktische Augenheilkunde, 1891, S. 237.
[7] Nordisk ophthalmologisk Tidsskrift, iv. S. 170.
[8] Inaugural Dissertation, 1873.
[9] Transactions of the American Ophthalmological Society, 1885.
[10] Ibidem, 1887.
[11] Royal London Ophthalmic Hospital Reports, vol. x.

Fig.1.

Fig.2.

In one instance Oliver[1] saw a blood-current in nearly all the vessels. During this stage of the affection an intermittent pulsatory entrance of blood into the central artery has been rarely found (Elschnig).[2]

These current-phenomena generally last but a brief time. They frequently give place to an increased fulness of the vessels, this continuing simultaneously with the appearance of *hemorrhages;* whereas, when there is merely an improvement in the filling of the vessels or when the vessels remain contracted, hemorrhagic extravasation rarely takes place. Fischer[3] found hemorrhages noted forty seven times in one hundred and fifty-five reported cases of embolism in the trunk of the central artery, these usually occurring in the vicinity of the disk and the macular region.

The red spot in the centre of the fovea may be due to a blood mass (Fuchs).[4] Hemorrhages usually appear from three to twelve days after the onset of the affection. In cases in which the vessels of the retina have for a time even shown good calibre, atrophy of the vessels, with a diminishing blood-supply, produces a pallid nerve-head, and an atrophic retina becomes noticeable.

After a varying period of time the retina itself, as a rule, regains its transparency. The vessels may become narrower, and their walls so thickened that finally merely narrow delicate threads remain; or the vessel walls may become so opaque as to appear as white- or yellowish-white-lined bands, in the middle of which thin threads of blood are evident. The arteries are affected to a greater degree than the veins. Most of the cases of so-called embolism of the central artery advance to complete or almost total blindness. When recovery of vision takes place it is often but partial. If the defect is central (giving rise to central scotomata), vision will be poor. Should central vision remain good, sight will be more or less useful. At times it has been observed that what was at first a darkening of the entire field may give place to a partial defect in the visual field. Thus a quadrant area or one of the lower or the upper half of the field may remain blind, the macula sharing to some extent in the loss of function. Quadrant or sectorial defects occur most frequently in embolism of the branch of the cerebral artery. A permanent central scotoma has been but rarely described (Eales,[5] Hirsch,[6] Jäsche[7]).

## II.—BRANCH EMBOLISM.

When the phenomenon depending upon an obstruction to the blood-supply presents itself in the area that is supplied by a branch of the cerebral artery, it is at the present time viewed, without exception, as being due

[1] Transactions of the American Ophthalmological Society, 1888.
[2] Archiv für Augenheilkunde, Band xxiv. S. 67.
[3] Ueber die Embolie der Arteria Centralis Retinæ, Leipzig, 1891.
[4] Lehrbuch der Augenheilkunde
[5] The Ophthalmic Review, 1882.
[6] Archiv für Augenheilkunde, Band xxxiii.
[7] Ibidem, vol. xxvii.

to embolism. In typical cases of occlusion of a branch of the central artery there is a primary defect in the visual field which it is not difficult to associate with an area which is supplied by a small vessel; or vision fails in both eyes, then returns first in one eye and afterwards in the other; in the latter, however, only in a portion of the field (Barkan).[1] When the upper half of the visual field is wanting, there is a constriction of the inferior branch. If the upper inner quadrant of the field is blind, there is a disturbance in the circulation in the lower outer branch. If the superior or the inferior temporal artery is affected, there will be a darkening of the corresponding quadrants of the field of vision, with more or less involvement of central vision. Central vision is not disturbed when the superior or the inferior temporal artery is obstructed, only when the macular twigs are obstructed. At times a white or a yellowish-white spot situated on the affected vessel has been assumed to be an embolus. Gray-white plugs have also been seen in the obstructed artery. Within the area of distribution of the changed vessels the veins in the beginning of the disturbance are somewhat contracted, but they soon become better filled and at times are abnormally broad.

When examined soon after the onset of the attack, a retinal opacity may be observed in the area of the distribution of the affected artery, similar to that which occurs in embolism of the trunk. *Especially interesting, however, is the fact that, in a vast majority of cases of embolism of a branch of the central artery, hemorrhages into the retina are absent or are present only in insignificant numbers.* Too little attention has been paid to this fact. The prevalence of the idea that numerous hemorrhages occur in the region of the occluded artery is probably owing to the theory that the blindness is due to hemorrhagic infarction. This, however, is not the case. In the accurately described cases of Saemisch,[2] Hirschmann,[3] Barkan,[4] Knapp[5] (three cases), Schön,[6] Nettleship,[7] Landesberg,[8] Hirschberg[9] (six cases), Mules,[10] Poulett,[11] Mittendorf,[12] Wadsworth,[13] Holden[14] (five cases), Tatham Thompson,[15] Perles[16] (four cases), Hirsch[17] (three cases), Haab,[18] Stuelp,[19] Stölting,[20]

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[1] Archiv für Augenheilkunde, Band iii. S. 175.
[2] Klinische Monatsblätter für Augenheilkunde, 1866. [3] Ibidem.
[4] Archiv für Augenheilkunde, Band iii. [5] Ibidem.
[6] Lehre vom Gesichtsfeld, S. 93.
[7] The Lancet, 1875.
[8] Archiv für Augenheilkunde, Band iv.
[9] Centralblatt für Augenheilkunde, 1885.
[10] Transactions of the Ophthalmological Society of the United Kingdom, 1888.
[11] Ibidem.
[12] Transactions of the American Ophthalmological Society, 1890. [13] Ibidem.
[14] Archives of Ophthalmology, vol. xxii.
[15] The Ophthalmic Review, 1891.
[16] Centralblatt für Augenheilkunde, 1891.
[17] Archiv für Augenheilkunde, Band xxxiii.
[18] Atlas der Ophthalmoskopie, 1895, Taf. 26.
[19] Centralblatt für Augenheilkunde, 1897.
[20] Archiv für Ophthalmologie, Band xliii. S. 306.

Jäsche,[1] and two cases seen by the author that have not yet been published, no trace of hemorrhage into the retina is mentioned as being present in any one. In four other cases seen by Hirschberg (*loco citato*) and Stölting (*loco citato*) merely faint traces of hemorrhage into the retina were present. The observation of Knapp[2] is cited as an instance of hemorrhagic infarct, but requires further examination. It is especially stated that a small venous branch lying within the area of the hemorrhagic infarct could not be traced back to the optic nerve, and the illustration shows this. It can be well assumed that in this case, which first came under observation three weeks after the beginning of the disturbance, the obstruction to the vein was primary, and the arterial change was secondary. In the discussion of thrombosis of the retinal veins, we shall see that this may be followed by a secondary affection of the arteries, which, under certain conditions, creates a certain picture which is similar to that given by Knapp. The case described by Landesberg (*loco citato*), under the designation of hemorrhagic infarct, does not exactly correspond to that condition, as the hemorrhages are not sufficiently numerous.

*In all the related cases of so-called branch embolism there was either an incomplete occlusion of the arterial branch so that no hemorrhagic infarct could occur, or the hemorrhagic infarct was not a result of obstruction to a branch of the retinal artery.*

The areas of distribution of the various branches of the retinal artery are separate, so that by occlusion of a branch the area that is supplied by the affected vessel remains blind. In this way it is proved (Hirsch)[3] that the blood-supply of the retina is divided into three independent regions: 1, The area of the superior principal branch; 2, the area of the principal inferior branch; 3, the area of the small arteries which come off from the papilla and go towards the macula lutea. Each of the principal areas may be eliminated without the macula suffering, but occasionally this region may also become blind,—*i.e.*, if it has received its blood-supply principally from the superior and the inferior main branches.

## CILIO-RETINAL VESSELS.

These vessels, when small, supply only the macula and occupy the position of the ordinary macular twigs. Occasionally they are of a larger size and supply a portion of the temporal half of the retina, they then being very rarely accompanied by a cilio-retinal vein. The frequency of their appearance, according to Lang and Barrett,[4] who examined forty-eight cases, is sixteen and seven-tenths per cent. According to Elschnig,[5] the condition was found in seven per cent. in one hundred and seventy persons who were examined.

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[1] Archiv für Augenheilkunde, Band xxvii. [2] Ibidem, vol. i.

[3] Ibidem, Band xxxvii.

[4] Royal London Ophthalmic Hospital Reports, 1888, vol. xii.

[5] Archiv für Augenheilkunde, Band xliv.

Only an anatomical study can determine whether these arteries come from the chorioid or from the central artery. Loring,[1] Nettleship,[2] Schleich,[3] Müller,[4] Nettleship,[5] Kuhnt,[6] Birnbacher,[7] le Plat,[8] Randall,[9] Goh,[10] and Elschnig[11] have all reported such investigations. Elschnig determined that they were of ciliary origin, and were derived from the scleral vessels (circlet of Zinn). As the circlet of Zinn contains only arteries, this explains why the vast majority of cilio-retinal vessels are arteries. Leber[12] also describes this important connection. Two to four, or even more (von Jäger), small branches of the short ciliary arteries enter the sclera in the vicinity of the optic nerve and there form a wreath which encircles the optic nerve. Numerous branches run to the chorioid and to the nerve and its sheaths. Corresponding veins do not exist. A less direct connection is formed between the vessels of the chorioid and the optic nerve by numerous small vessels that, entering the optic nerve through the chorioidal ring, are continued directly as a free capillary net-work in the great meshes of the intraocular end of the optic nerve.

These cilio-retinal vessels do not establish any marked anastomosis between the ciliary and the central vessel system of the retina. In those cases of obstruction of the central artery in which a cilio-retinal vessel is present, the retina undergoes the usual necrosis, and only the small area that is supplied by the cilio-retinal artery remains intact. The writer has seen a case (not yet published) in which such an artery supplied in part the macular region; and its obstruction, which was not assumed to be of embolic nature, gave rise to a scotoma that corresponded to the circumscribed opacity of the part of the retina that was supplied by the vessel. In some of the cases seen by Benson,[13] Birnbacher,[14] Mittendorf,[15] and Wadsworth,[16] one or more cilio-retinal vessels supplied the intact area. In the remaining fifteen cases these were of the macular type not possessing the character of cilio-retinal vessels, which remained free from obstruction. In the case reported by Laqueur,[17] and in the sixteen cases collected by him, it

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[1] Archiv für Augenheilkunde, Band ii.
[2] Royal London Ophthalmic Hospital Reports, vols. vii. and ix.
[3] Mittheilungen aus der ophthalmischen Klinik in Tübingen, Heft 1.
[4] Archiv für Augenheilkunde, Band iv.
[5] Royal London Ophthalmic Hospital Reports, vol. ix.
[6] Bericht der ophthalmologischen Gesellschaft, Heidelberg, 1881.
[7] Archiv für Augenheilkunde, Band xv.
[8] Annales d'Oculistique, 1885.
[9] Transactions of the American Ophthalmological Society, vol. iv.
[10] Archiv für Augenheilkunde, Band xliii. S. 148.
[11] Ibidem, vol. xliv.
[12] Handbuch der gesammten Augenheilkunde, Band ii. S. 305.
[13] Royal London Ophthalmic Hospital Reports, 1882, p. 336.
[14] Centralblatt für praktische Augenheilkunde, 1883, S. 207.
[15] Transactions of the American Ophthalmological Society, 1882.
[16] Ibidem, 1890 and 1896.
[17] Archiv für Augenheilkunde, 1895, Band xxx. S. 75.

is shown that the visual fields of these eyes were small, oval, triangular, or oblong, and extended from ten to twenty degrees in their horizontal diameters. In those cases in which the non-obstructed artery has the appearance of being a branch of the central artery, it must be assumed that the vessel was given off behind the point of obstruction in the central artery. The writer suspects, however, that in these cases the vessels were cilio-retinal in character.

### OPTICO-CILIARY VESSELS.

This peculiar and rare communication between the retinal vessel system and that of the ciliary vessels has been observed by Elschnig,[1] Rumschewitsch,[2] Nickels,[3] Lawford,[4] Oeller,[5] Kuhnt,[6] and Salzmann.[7] Such vessels possess no significance in the study of obstruction of the central artery, but simply show another variety of anastomosis that exists between the ciliary and retinal or optic nerve-vessel system.

### CRITICAL CONSIDERATION OF THE PHENOMENA ARISING FROM OBSTRUCTION OF THE CENTRAL ARTERY.

It is incorrect in these cases to make always the stereotyped diagnosis of embolism, for clinical observation, as has already been seen, often fails to reveal a source of origin for the embolus. In the writer's opinion, the existence of a heart-murmur is not sufficient proof, but an exhaustive systematic examination must demonstrate a *vitium cordis* to produce an embolism. Further, Sperling, who has collected the results of the autopsies of the Virchow Institute during the years 1868–70, reports seventy-six cases in which emboli were found in the body, the brain being affected only fifteen times, while the kidneys were involved in fifty-seven cases. The heart must again be considered relatively: chronic endocarditis often occurs as the result of nephritis. If, in a case of obstruction of the central artery, a valvular disease is found, it does not prove that the obstruction was due to an embolus, for it may be that the primary disease existing in the kidneys also caused sclerosis of the central artery. The aorta and the carotids are considered the seat of origin for embolus in the central artery in those cases in which atheroma of the peripheral arteries is demonstrable. In the judgment of the writer, however, it would be more rational to assume that the central artery has also undergone a sclerosis, and that this localized condition has given rise to an obstruction of the local vessel. The same may be said of aneurism of the aorta and the carotids. Syphilitic disease of the vessel walls has attained a much wider significance through the investiga-

[1] Archiv für Augenheilkunde, Band xviii. S. 41.
[2] Klinische Monatsblätter für Augenheilkunde, 1889, S. 41.
[3] Ibidem.
[4] Transactions of the Ophthalmological Society of the United Kingdom, 1895, p. 95.
[5] Atlas der Ophthalmoskopie.
[6] Bericht der ophthalmologischen Gesellschaft, Heidelberg, 1881.
[7] Archiv für Ophthalmologie, Band xxxix.

tions of Backhouse.[1] The frequent simultaneous occurrence of aneurism of the aorta with this affection makes it extremely probable that mesarteritis is the cause of aortic aneurism in syphilis. In fifty-nine syphilitic cases that came to autopsy at Kiel, Phillips[2] found that over twenty-seven per cent. of syphilitic mesarteritis of the aorta was present. In an examination of one hundred and one cases of aneurism of the aorta, Malmsten[3] has shown the presence of syphilis in eighty per cent. Puppe[4] found syphilis in seven cases out of sixteen of aortic aneurism.

The origin of an embolus from a venous thrombus is not so simple as is at times taught. In the opinion of the writer, it is difficult to attribute to emboli cases in which the picture of embolism of the central artery is present in both eyes. Jäger,[5] Landesberg,[6] Page,[7] Haase,[8] Vossius,[9] and Alexander[10] have all published such cases. The prodromal attacks of blindness which often occur, and at times appear in both eyes, make it difficult to accept the theory of embolism. These attacks are probably due to the fact that one or both of the central arteries have become narrowed by disease of the walls, and a transient lowering of cardiac force has so encumbered the circulation that it has temporarily suspended the function of the retina (Loring).[11] This theory explains those cases in which digitalis has undoubtedly acted unfavorably upon the eye-affection (Schneller,[12] Mittendorf,[13] and Alexander[14]).

The phenomenon that we have so often noticed in our own patients is that soon after the occurrence of a total or a partial blindness a moderate or even good diameter of the retinal vessels, with the evidence of distinct circulation, appears, and in not a few cases the function of the retina becomes partially or entirely restored; this speaks further against the idea of a true embolus.

It is impossible that the obstruction of the central artery can be compensated for, in any degree, by collateral circulation or anastomosis. Schnabel and Sachs[15] assume that the embolus is smaller than the lumen of the vessel, and that localized contraction of the central artery holds the embolus fast; it adheres to the walls and permits a varying amount of

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[1] Beiträge zur pathologischen Anatomie, Leipzig, 1897, Band xxii. S. 417.
[2] Statistik der Envorbenen Syphilis, Inaugural Dissertation, Kiel, 1896.
[3] Aorta Aneurysmens Etiologie, Stockholm.
[4] Deutsche medicinische Wochenschrift, 1895.
[5] Wiener medizinische Presse, 1868.
[6] Archiv für Augenheilkunde, Band iv. S. 109.
[7] American Journal of the Medical Sciences, 1874.
[8] Archiv für Augenheilkunde, x.
[9] Klinische Monatsblätter für Augenheilkunde, 1883, S. 298.
[10] Bericht der ophthalmologischen Gesellschaft, Heidelberg, 1896, S. 258.
[11] American Journal of the Medical Sciences, vol. lxvii, 1874.
[12] Archiv für Ophthalmologie, viii.
[13] Transactions of the American Ophthalmological Society, 1882, p. 411.
[14] Loco citato.
[15] Archiv für Augenheilkunde, Band xv.

blood to pass by. This, in the opinion of the writer, is to be doubted, although Herrnheiser[1] has observed, experimentally, this condition in embolism of the retinal vessels in rabbits. Wagenmann[2] also attempts to explain how the circulation can be re-established, but as yet anatomical proof is wanting.

There are other clinical symptoms which make us hesitate in assuming that obstruction of the central artery is always due to embolism. Albuminuria has been proved to have been present in quite a large percentage of the published cases, and, according to Fischer, sclerosis occurs in a great proportion of these cases; therefore, sclerosis may be very pronounced in the retinal vessels, as has been seen recently by the writer in a case of so-called embolism. Raehlmann[3] found in a supposed case of embolism complete obliteration of the vessels by endarteritis. An analogous case of bilateral blindness and chronic interstitial nephritis, in which the arteries of the retina were nearly bloodless, is described by Sodato.[4] Among such other causes may be mentioned quinine intoxication, in which Gruening,[5] Buller,[6] de Bono,[7] and Berger[8] have all observed phenomena that are similar to those found in embolism. Similar pictures have been seen by Knapp[9] in erysipelas, by Carl,[10] Emrys-Jones,[11] and once by Knapp[12] after a knife-stab of the orbit. Finally, there is a group of cases communicated by Ewars,[13] Barkan,[14] Gunn,[15] and Batten,[16] in which no other pathological process, except anæmia and sclerosis, could be found.

According to the stand-point of the investigator in regard to the controversy over this question he has declared the obstructing mass to be an embolus or a thrombus, and, in the opinion of the writer, in not a single instance, not even in which it was very likely to be an embolus, has the proof of embolic nature been at all convincing. It is not a simple matter to give an anatomical proof of an embolus, and in order to show conclusively the existence of an embolic occlusion of the central artery, the investigator must not only be fortunate in obtaining the material at the proper time, but he must also, as B. Ribbert points out in his Text-book of

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[1] Klinische Monatsblätter für Augenheilkunde, 1895, S. 315.
[2] Archiv für Ophthalmologie, Band xxxviii. S. 3.
[3] Vortschritte der Medicin, 1889, S. 929.
[4] Archivio di Ottalmologia, 1895, vol. ii. p. 361.
[5] Archiv für Augenheilkunde, Band ii.
[6] Transactions of the American Ophthalmological Society, 1881.
[7] Archivio di Ottalmologia, vol. ii.
[8] Les Maladies des Yeux, Paris, 1892.
[9] Archiv für Augenheilkunde, Band xiv. S. 269.
[10] Klinische Monatsblätter für Augenheilkunde, 1884, S. 118.
[11] British Medical Journal, 1884, vol. i. p. 312.
[12] Archiv für Ophthalmologie, xiv., I., S. 218.
[13] Zweiter Jahresbericht, 1872.
[14] Loco citato.
[15] Transactions of the Ophthalmological Society of the United Kingdom, 1895.
[16] Ibidem.

Pathological Histology (Bonn, 1896), employ all known technical aids. The *orcein stain*, however, aids greatly in such a study of the vessel, as it well differentiates the elastic elements of the structure of the walls. The anatomical diagnosis of obstruction of the vessel does not lie entirely between the question of an embolus or a thrombus, but also includes the question of endarteritis obliterans. In the writer's anatomical studies this last condition seems to present itself not infrequently, and in such a manner as to contract or completely close the affected vessel in isolated places, so that an investigator could easily confound the condition with an embolism or a thrombosis. Some of the illustrations given (Elschnig, Marple, Wagenmann) force the reader to the assumption of an obliterating endarteritis as the cause. The central artery of the retina and its branches appear to have a predilection for such obliterating diseases, possibly on account of the sharp bending of the vessels at the point of ramification upon the papilla, the intra-ocular pressure, and the movements that the intra-ocular end of the optic nerve undergoes with the movements of the eye.

Perhaps these factors favor not only obliterating processes, but are also productive of such changes in the vessels that have an injurious effect upon the intima, so that, as a result, *thrombosis* develops when, for any reason, a lowering of the blood-pressure follows. A very instructive illustration is afforded by the so-called embolism of the central artery in its relation to glaucoma. Here the vessel-changes are found very frequently (Edmunds and Brailey),[1] especially in the hemorrhagic type (Pagenstecher,[2] Deutschmann,[3] Hache,[4] Panas,[5] Stölting,[6] and others). If it be desired to study vessel sclerosis and obliterating endarteritis in the eye, it cannot be better done than in examination of an eye that has been enucleated for hemorrhagic glaucoma. Endarteritis may even lead to aneurismal formations in the glaucoma (Liouville,[7] Panas,[8] Hulke,[9] Edmunds and Brailey,[10] Ponsett and Wagenmann[11]). The anatomical studies of Ridley[12] show that thrombosis of the central artery can also take place in glaucoma.

The fact must not be overlooked that in the sixteen cases of so-called embolism of the central artery of the retina that have been examined anatomically, five were enucleated on account of glaucoma, which permits of the assumption that it was possible, without emboli, to have had an oc-

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[1] Royal London Ophthalmic Hospital Reports, x. p. 2.
[2] Atlas der pathologische Anatomie.
[3] Archiv für Ophthalmologie, Band xxv. S. 3, 163.
[4] Thèse de Paris, 1874.
[5] Traité des Maladies des Yeux, Paris, 1894.
[6] Archiv für Ophthalmologie, Band lxiii. 2, S. 308.
[7] Comptes-rendus de la Société de Biologie, 1868, Thèse de Paris, 1870.
[8] Leçons clinique de Ophthalmologie, 878.
[9] Practical Treatise on the Use of the Ophthalmoscope.
[10] Loco citato.
[11] Bericht der ophthalmologischen Gesellschaft, Heidelberg, 1897, S. 154.
[12] Royal London Ophthalmic Hospital Reports, vol. xiv., 1895, p. 264.

clusion of the central artery, either by a thrombosis or an endarteritic obliteration of the vessels. It may be said that if this supposition is correct, then obstruction of the central artery and its branches should be observed oftener in glaucoma, though probably it is present more frequently than has been supposed. A very scanty retinal blood-supply is usually seen in glaucoma, and at times a blindness which cannot be rightly explained unless it is assumed that a contraction of the central artery or its branches has led to functional death of the optic nerve and retina, may be noticed. One way, of course, is to accept the view of Leber[1] that this condition has been produced by a spasm of the vessel, but as yet this condition has seldom been found with any degree of certainty in the human eye. Priestley Smith,[2] Galezowski,[3] and others have also considered vessel-spasm in explaining the clinical picture of thrombotic obstruction of the central artery. Raynaud[4] and others have described spasm of the retinal artery as occurring ophthalmoscopically in Raynaud's disease. Knies[5] has seen the vessels of the retina become narrowed immediately before an epileptic seizure. His observation has been confirmed by de Bono and Dotto.[6] Schnabel[7] attributes to spasm-ischæmia a very great contraction of the retinal arteries with a corresponding dilatation of the veins occurring in the stage of chill in intermittent fever. It is uncertain whether the cases of retinal ischæmia of A. Graefe,[8] Rothmund,[9] Knapp,[10] and others were due to arterial spasm.

On the other hand, the rapidly passing localized vessel-spasm which Benson[11] saw in his case cannot well be ascribed to other cause than such a condition, unless it be assumed that visible circulation of the blood with interruption of the blood column simulates this remarkable case of peristalsis. A thirty-two-year-old man, without heart- or kidney-disease, who had had malaria and rheumatism and who was suffering from tobacco-amblyopia, had been having attacks of total or partial blindness, lasting but a few minutes at a time for four years' time. Latterly only the right eye was affected. During the attacks it was noted that sectors of arterial branches became empty and pushed their way towards the periphery of the fundus; and this, at times, in a pulsatory manner. To the writer, the fact that pressure upon the left eye induced quicker intermittent filling and emptying of the arterial terminals is important, as showing that the blood-

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[1] Bericht der ophthalmologischen Gesellschaft, Heidelberg, 1897, S. 162.
[2] British Medical Journal, 1874.
[3] Recueil d'Ophthalmologie, 1882 and 1892, and Traité d'Ophthalmologie.
[4] Archives générale de Médecine, 1874.
[5] Bericht der ophthalmologischen Gesellschaft, Heidelberg, 1877.
[6] Archivio di Ottalmologia, 1894, p. 238.
[7] Archiv für Augenheilkunde, 1885, S. 377.
[8] Archiv für Ophthalmologie, Band viii.
[9] Klinische Monatsblätter für Augenheilkunde, iv. S. 106.
[10] Archiv für Augenheilkunde, Band v.
[11] Transactions of the International Ophthalmological Congress, Edinburgh, 1894, p. 81.

pressure in the left retinal arteries was less than that in the right. It permits the consideration of as yet unknown factors in the weakening of the blood-current, from causes that are situated within or without the eye. The case of Wagenmann may also permit of a similar assumption. *The advancing contraction* observed by Benson alone in the vessels is most surprising, for such peristaltic spasmodic movements have not been previously observed in blood-vessels.

The assumption of a vessel-spasm would render great service in explaining the prodromal periodic blinding and especially the filling of the retinal vessels that is seen immediately after the blinding. The presence of an embolus would be unnecessary to explain the clinical symptoms, as they could be understood as follows. As a result of disease of the vessel-walls, a more or less general narrowing of the lumen of the vessels occurs, but in such a way as not to interrupt the retinal function. If now, as a vessel-spasm takes place, the symptoms are readily explained, and if thrombosis of the central artery occurs, this would explain the permanent obstruction of the vessel. Vessel-cramp without thrombosis, continuing for several hours' time, might destroy the retinal function. Leber[1] comments on the feasibility of this theory, especially in connection with Wagenmann's communication, and suggests further study of this subject, in which opinion the writer concurs. Positive proof of vessel-spasm in either normal or diseased retinal vessels is, however, largely wanting, and the writer is inclined to assume that other conditions must at times be taken into consideration. The transient prodromal attacks of blindness may be explained by supposing that when the arteries are narrowed by endarteritis, etc., a slight sinking of the blood-pressure is sufficient to interfere with the nourishment of the retina. Perhaps narrowing of the vessels arising from cellular thickening of the walls may disappear and the blood-current be again set free, as the writer has recently observed in a case in which he considered the contraction due to a change in the walls that disappeared without any therapeutic measure of importance. There are changes in the retinal vessels which still baffle our comprehension.

All these various conditions and theories must be taken into consideration in diagnosing the character of the obstruction of the central retinal artery.

Prognosis is always grave, especially if the cause be embolic in type. Treatment, which will vary according to the nature of the arterial obstruction, may be divided into general and local. The former will be directed toward heart failure, syphilis, etc., and will include the use of digitalis, mercury, iodide of potassium, nitrite of amyl, and the prone position of the patient. Digitalis must not be neglected, even if the presence of an embolism is suspected, for if the clot be attached to the walls, this drug is of service, and if it completely fills the artery, digitalis will not greatly increase any damage.

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[1] Loco citato.

Local treatment will avail most when the obstruction is not embolic in character; although iridectomy is of no value in most cases. Still, however, a sclerotomy or puncture of the sclera can be recommended in recent cases of thrombosis, as has been advocated by Mauthner,[1] who might also be considered the pioneer in the so-called massage treatment, as he recommends deep pressure upon the globe in quite recent cases of embolism. This he does in order that in case the embolus projects but a short distance into the central artery and blood can still enter in small quantity, this procedure will hinder the further entrance of blood into the interior of the eye, and thus by damming the arterial blood prevent the complete entrance of the embolus into the central artery, from which point it would be swept farther on. If pressure fails, a sclerotomy should be performed, so that by *vis a tergo* the embolus can be driven from the trunk into a branch. This view, according to the writer, appears to apply more to soft emboli.

True massage was first applied by Wood-White,[2] with good results, in a case of typical embolism with blindness, resulting in recovery of normal vision. Such a result has been confirmed by Mules, Hirschberg, Hilbert, Fischer, and Gifford. The writer is convinced of the assertion of Schnabel and Sachs,[3] that pressure-experiments demonstrate that by massage the circulation of the retina can be increased, and that when the pressure is discontinued the pupil becomes slightly red, previously invisible vessels become demonstrable, and stretched arteries assume a serpentine form. When these authorities further assume that by this means a recent soft, incomplete embolus can be moved or even broken up and thus rendered harmless, the writer would still accept their dictum, only he would say, *not embolus, but thrombus.*

## DISTURBANCE OF THE CIRCULATION WITHIN THE AREA OF THE CENTRAL VEIN OF THE RETINA AND ITS BRANCHES, OCCASIONED BY THROMBOSIS.

Michel[4] distinguishes three grades of intensity in this affection, depending upon (1) complete obstruction, (2) incomplete obstruction, and (3) thrombosis which is so slight that there is a stagnation of the blood of the venous system of the retina.

According to Michel, the disease pursues three different courses in conformity with the pathologico-anatomical conditions. In the first type, in which the thrombus becomes organized and permanent, the hemorrhages are at first partly absorbed and the borders of the papilla, by reason of hemorrhagic coloration, glisten somewhat. Soon, however, the vision grows worse again, accompanied with fresh hemorrhages. These relapses recur

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[1] Wiener medizinische Blätter, vi., 1883.
[2] The Ophthalmic Review, 1882.
[3] Archiv für Ophthalmologie, 1878, iii.
[4] See Schöbl's article in this System, vol. iii. p 442.

until the retina, together with the vitreous, becomes hazy and vision practically abolished. In the second grade, the hemorrhages gradually disappear, being changed into yellowish-white spots in such a way that in the beginning the light color appears in the centre of the hemorrhage, the vision gradually improving, sometimes to a considerable degree. In the third degree of intensity, all ophthalmoscopic changes, except the dilatation of the veins and the whitish color of the papilla, may, in varying periods of time, disappear.

According to Michel, these three courses correspond to (1) complete occlusion through organization of the clot; (2) partial re-establishment of the lumen by molecular destruction of the thrombus, which becomes partly organized; and (3) complete re-establishment of the permeability of the vein. Michel has never seen increase of tension. Anatomically, the organization of the thrombus was largely incorporated with the inner wall of the central vein, it being only about one and a half millimetres long and about six millimetres removed from the entrance of the optic nerve into the eyeball.

Thrombosis of the central vein may occur as a complication in glaucoma, pernicious anæmia, and diabetes. It occurs metastatically in thrombosis of the orbital veins of septic or marasmic origin simultaneously with the appearance of thrombosis of one or more of the sinuses of the brain. Angelucci[1] has published three cases of thrombosis of the central vein in which retinal hemorrhages or greatly distended veins were not present. Von Zehender[2] explains the absence of hemorrhage in Angelucci's first case by supposing that the venous thrombus compressed the central artery and thus diminished the blood-supply to the retina, while Michel disputes that it was a case of thrombosis. Certainly the anatomical proofs in all three of Angelucci's cases do not seem free from criticism.

The case observed by the writer and published by his assistant (Turk) agrees fairly with the findings of Michel. A very small thrombus that completely obstructed the vein was situated in the posterior part of the lamina cribrosa. The distention of the larger veins and the enormous infiltration of the retinal tissue that were found in the microscopic preparations of this case are shown in Figs. 5 and 6. The section shown in Fig. 5 is taken from the vicinity of the papilla, and that of Fig. 6, somewhat farther away. In the latter the formation of spaces through œdema is very striking.

The theory of marasmic thrombosis receives confirmation from a case seen by Goh (Case II.). In a reported instance he considers the venous obstruction as marantic, notwithstanding the septic nature of the disease, owing to its being bilateral; whereas, with Axenfeld, he considers unilateral thrombosis as described by Michel to be dependent on local disease of the vessels.

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[1] Klinische Monatsblätter für Augenheilkunde, 1878, S. 443, und 1879, S. 15.

[2] Bericht der ophthalmologischen Gesellschaft, Heidelberg, 1878, S. 182.


Fig. 5.


Fig. 6.


Fig. 7.

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Lith. Anstalt F. Reichhold, München.

While the relation of the process to increased intra-ocular pressure remains entirely in the background in Michel's presentation, yet, in the opinion of the writer, the cases that have been studied anatomically by Weinbaum, Schnabel, Wagenmann, Würdemann, and Purtscher show a very important relation to glaucoma, as has been seen in the consideration of embolism of the central artery.

What is termed thrombosis of the central vein leads occasionally to glaucoma. Like the artery, the central vein seems to have a predilection for degenerative changes. What appears, however, to be of the most importance in the production of the affection is that, simultaneously with general or local arterio-sclerosis, the circulation in the eye is disturbed, and to this is added a weak cardiac action that favors venous thrombosis. Without simultaneous arterio-sclerosis thrombosis of the central vein seems but rarely to occur.

The writer, from his own experience in sixty thousand case-histories, would add the following as his conclusions: Typical cases of this disease, in which albuminuria can be excluded, are of infrequent occurrence. Heart-disease seems to play a favoring *rôle*, probably because it produces early arterio-sclerosis. Almost all of the writer's cases have been engaged at hard work all their lives; the vast majority occurring in the peasant classes, who work so much in a stooping posture. The affection is rarely bilateral, possibly because, after the loss of the first eye, the patient takes better care of himself. Differing from the experience of Michel, the writer has more frequently met with prodromal obscurations and phosphenes; certainly, however, the disturbances of vision usually appear without these signs, and often occur at night.

Obstruction of the branch of the artery takes place, on the whole, earlier in life than that of the trunk,—*i e.*, between forty and fifty years. One or more venous branches may be obstructed, so that hemorrhage, opacity of the retina, congestion of the veins, and the ever-present white splotches may all appear only in the region of a venous branch. (Fig. 3.)

The venous obstruction that is due to such thrombosis can lead to secondary disease in the artery corresponding to the affected vein. This secondary disease has been carefully studied by Thoma. In his case dilated collateral vessels carrying venous blood towards the papilla were visible towards the macula. In this region a dilated arterial twig could be seen. This case gave an important insight into the nature of the white degenerative areas. Although the patient did not have albuminuria, such areas were to be seen in the area of the obstructed vein, especially towards the macula lutea, these spreading out until, within three days after the disturbance of vision, they had reached the middle of the fundus. Not a single additional area of white degeneration appeared, nor did the hemorrhages become converted into such. The writer believes that, in general, these retinal hemorrhages do not change into white areas, and that the degenerative areas have nothing directly to do with the hemorrhages, but that they are formed of

groups of fat-granule cells (phagocytes) that take up the *débris* of the destroyed retinal elements. The white areas are also expressions of a serious disturbance of nutrition of the retinal tissues from abnormal conditions of the blood or the vessels, but in no way are they the direct product of hemorrhagic degeneration, as seen in cases of uncomplicated traumatic retinal hemorrhage in which such areas are not present. These areas have a more serious prognostic import than the hemorrhages that are seen in albuminuric retinal degeneration.

The writer would not venture to answer the question whether in this process a thrombosis is being solely dealt with, and whether it is right to attribute it to marasmic influences, for the conditions here met with are very complicated. It appears possible, however, that in a case in which all of the vessels are normal nothing could result from obstruction of the central vein except stasis and œdema. This assumption, however, requires further study.

In cases of the first and the second grade, in which the phenomena are very pronounced (Fig. 4), the intermittent flow of blood into the arteries upon the disk resumes its normal condition when pressure is made upon the globe. Consequently, the circulation in the retinal vessels must be but little altered, despite the marked retinal changes. In cases of the second grade the writer has been able to induce an immediate, intermittent arterial flow with a simultaneous emptying of the veins upon the papilla and its vicinity by pressure on the globe. In this type the blood finds a ready exit through the vein, thus proving the permeability of the vessel. The degree of destruction of the retina and the number of hemorrhages found have but little connection with the degree of obstruction of the vein, but more with the perviousness of the retinal vessels.

In cases in which there is still ordinary circulation a considerable number of hemorrhages may appear, and likewise abnormal distention of the venous branches may take place. This is also the result of a marked diminution in the elasticity of the walls of the veins. In the future anatomical research must be directed particularly towards the condition of the vessels of the retina, and here also proper staining will bring about the desired solution of the problem.

The assumption that obstruction of the central vein in this condition is of a marasmic nature applies only to a small number of cases or the affection would more frequently be bilateral. By preference it affects individuals who are quite strong, and who in many cases have never had a day's illness, but have performed hard manual labor, and who have often overtaxed their hearts.

With the above clinical picture thrombosis, or a more or less marked degree of obstruction of the central vein from other causes, will have to be assumed as the cause until further anatomical study is presented. Leber[1]

[1] Handbuch der gesammten Augenheilkunde, Band v.

Fig.3.

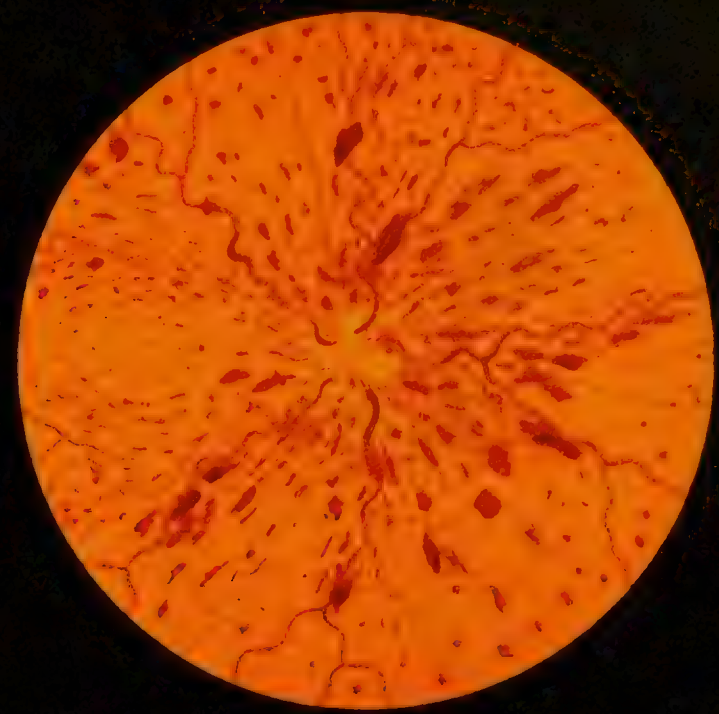

Fig.4.

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and Wagenmann[1] believe that multiple emboli in the branches of the retinal artery can provoke the same ophthalmoscopic picture as thrombosis of the central vein by the resulting hemorrhagic infarcts, and this view is apparently supported by the histological study of a case seen by the latter author. The writer doubts this, for the reasons given in speaking of branch-emboli. For it was there shown that obstruction of arterial branches does not produce hemorrhagic infarcts in the retina, and the case of Knapp,[2] which is always cited as one of hemorrhagic infarct of the retina, may, as above stated, be described as one of obstruction of a venous branch with secondary arterial degeneration. It is true that the artery corresponding to the obstructed vein did not show white walls, but the affection had not progressed as far in three weeks' time as in the case described by the writer, in which twelve months had elapsed.

In making the diagnosis the question of the presence of albuminuria is very important. So-called albuminuric retinitis can present a similar picture, and the question is pertinent whether in this very grave pathologic process thrombosis or obstruction of the trunk or of a branch of the central vein may not play a certain secondary part. As albuminuric retinitis may in the beginning affect but one eye, it is important in all cases that resemble thrombosis to examine the urine. When the affection occurs in the young syphilis must be suspected. For instance, the writer has seen the appearances of thrombosis of the second grade in the right eye of a patient twenty-four years of age. Although the vision equalled but one-third of normal, the symptoms disappeared and all of the functions were re established within three weeks' time from the time of the use of inunctions of mercury. A moderate proptosis, the result of a slight involvement of the orbit, disappeared simultaneously.

The writer can confirm the observation of Michel, as quoted above, that symptoms of thrombosis may result from anæmia, having seen it, not in pernicious anæmia, but in a case of intense anæmia of pregnancy. The pronounced changes, distention of the veins, numerous hemorrhages, white splotches, etc., all disappeared spontaneously three weeks after confinement, vision again becoming normal.

Thrombotic retinal affections are seen as complications of inflammatory, and especially infectious, processes in the orbit, whether they are caused by erysipelas, meningitis, thrombosis of the cerebral sinuses, or in any other manner. In this type of the affection it is difficult to determine how much is to be attributed to thrombosis of the retinal veins, and how much is the result of thrombosis of the central arteries. This is likewise the case in orbital phlegmon. In all of these cases, not only is thrombosis being considered, but phlebitis. Here the disease of the veins is an accompanying symptom of the inflammatory process, and does not concern the question.

As a rule, prognosis is unfavorable for the affected eye, but usually it

[1] Archiv für Ophthalmologie, Band xxxviii.

[2] Loco citato.

is good for the unaffected organ. Quite often, however, the afflicted eye acquires more or less, and at times a normal degree of vision. Much depends whether the macula lutea is involved. Numerous whitish dots in this region are especially ominous. Nevertheless, the writer has seen normal vision regained in spite of such conditions.

Treatment is ordinarily quite valueless, and in cases in which improvement occurs it is usually independent of anything that has been done. This result follows from the nature of the affection. The writer has occasionally seen evident benefit from the energetic use of digitalis, massage, and nitrite of amyl, but not in the above-described case, in which an obstructed section of the vein was plainly visible, and of a type in which it would be presumable that it would be influenced by massage.

The value of treatment lies in prophylaxis. Elderly people, who perform hard bodily labor, must be counselled to discontinue such work, and especially to refrain from prolonged stooping. This advice is particularly applicable to cases in which there is disease of the heart or the vessels. Those cases that have had one eye affected—having learned from experience—usually follow the advice, and, as a rule, save at least the one organ. Where there is the slightest suspicion of syphilis the patient should be placed under the influence of mercury and the iodide of potassium.

## DISTURBANCES OF THE CIRCULATION IN THE AREAS OF DISTRIBUTION OF THE POSTERIOR CILIARY ARTERIES.

As has been seen in the previous sections that many of the disturbances of the retinal blood-vessel system have been insufficiently investigated, it will be found that this applies in a still more marked degree to the post-ciliary system. Wagenmann[1] has proved that the disturbances in this area have great significance for the entire eye, especially for the retina, he finding that all of the elements in the portions of the retina that have been deprived of chorioidal circulation have rapidly degenerated. From investigations upon rabbits he determined that the entire retina became cloudy after division of the long ciliary and a portion of the short post-ciliary arteries, this being accompanied by a diffuse parenchymatous haze of the cornea. The lens became rapidly opaque, and its elements underwent degeneration. These results obtained in rabbits cannot, however, be referred to man without modification. The solution of this question in a great part belongs to the future.

The equally important question whether in man the individual ramifications of the post-ciliary artery communicate with one another or whether they constitute so many end-arteries is still unanswered. From the researches of Wagenmann and Segrist[2] it would appear that in rabbits they are end-arteries, supplying a certain area exclusively. Leber[3] found that the

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[1] Archiv für Ophthalmologie, Band xxxvi.

[2] Mittheilungen aus kliniken und medicinischen Instituten der Schweiz, 1895, Heft 9.

[3] Handbuch der gesammten Augenheilkunde, Band v.

capillary distributions of neighboring chorioidal vessels in man were in many ways associated. If it is assumed that the same relations exist in man as in rabbits, then an interference with the flow of blood in the post-ciliary arteries endangers to a greater or a less degree the entire retina. For instance, if obliterating disease of the post-ciliary vessels should occur, the retina would share in the disturbance, and possibly the arterio-sclerosis that is seen in contracted kidney (Duke Carl Theodor[1]) would be found in the chorioidal vessels. This would explain in part the degenerative changes that occur in the retina. Senile sclerosis of the chorioidal vessels produces disturbances of nutrition similar to those which arise from albuminuria. Hemorrhages, white degenerative areas, so-called thrombosis of the central vein, etc., produced by disturbances of nutrition, and in which the retina and its vessels suffer, can be seen as the results of sclerosis of the chorioidal vessels. It can be assumed that the numerous hemorrhages and the abnormal fulness of the vein have been caused by disturbance of the nutrition, corresponding in degree to the acuteness of the sclerotic affection of the chorioidal vessel. Therefore, the hypothesis of a thrombosis of the central vein can be dispensed with, and, without this theory, those cases in which the permeability of the central vein by the pressure test is proved are explainable.

Specific disease of the vessels can affect also those of the post-ciliary region, and, from resulting constriction and obstruction, endanger to a marked degree the integrity of the system. Changes induced by hereditary syphilis in the eye-ground may consist in a specific degeneration of the vessels in the chorioid.

In the future, in all pathological and anatomical investigations of the eyeball, examination must be made for accompanying disease of the post-ciliary vessels.

## DISEASE OF THE WALLS OF THE OCULAR VESSELS.

A consideration of the diseases of the vessel-walls in general shows that, as a result, the changes occurring in the tissues of the eye are as important in their frequency and significance as spontaneous extravasations of blood are. The patches of white degeneration that appear in the retina soon after hemorrhage seem to be more the result of poor nourishment of the tissues, resulting from the diseased vessels, than from the presence of the blood-clots. In simple traumatic hemorrhage such areas of degeneration scarcely ever occur. As a further consequence of disease of the vessel-walls aneurismal formations, which are demonstrable only microscopically, may be seen, these being associated with new blood-vessel formations in the retina and vitreous humor. In the pathological anatomy of the diseases of the blood-vessel walls there still exists a wide gap. Often it is only acci-

[1] Ein Beitrag zur pathologischen Anatomie des Auges bei Nierenleider, Wiesbaden, 1881.

dentally possible to obtain the material for investigation at the proper time. With the ophthalmoscope the true pathological changes are only to be discerned imperfectly, the microscope being the true interpreter.

## SENILE ANGIO-SCLEROSIS.

This affection plays a most destructive part in diseases of vessels of the eye. Here, as in other portions of the body, it may appear either in the diffuse or the circumscribed form. From the publications of Thoma and his pupils it would seem that the internal carotid artery and its area of distribution are especially prone to attacks by these degenerative processes, so that it is natural to suppose that these retinal changes are of special importance in the study of the pathology of the brain. It is well, however, to sound a note of warning as to the precept that the retina is to be considered a portion of the brain. The most important work in support of such a view has still to be performed, and must be done with the microscope, as with the ophthalmoscope but little is to be relatively seen.

The relationship existing between arterio-sclerosis and phlebo-sclerosis, as pointed out by the writer and his students, is observable. Raehlmann[1] has observed ophthalmoscopically that not only arterial but venous senile degenerations occur in the retina. It has been known for a long time, however, that in the ophthalmoscopic picture the changes in the walls of both the retinal and the chorioidal vessels are difficult to observe properly. Particularly is this true of angio-senile sclerosis, for in the majority of cases of this affection the existence of thickening of the walls of the retinal vessels can be merely inferred by a narrowing of the blood-columns. In some of the cases the changes in the walls of the vessels can be seen only in the form of gray or white streaks which bound the sides of the blood-columns or cover it in places. Very frequently it is impossible to determine whether the thickening of the walls of the vessels is the result of atheroma that may have resulted from acquired or hereditary syphilis, albuminuria, diabetes, leukæmia, etc. This degeneration resembles the vessel-atrophy that is seen after inflammation of the nerve and retina. It is often impossible to determine microscopically whether a degeneration of the vessels is syphilitic or senile in character. Naturally, therefore, it becomes even more difficult to prove in the ophthalmological picture. In cases in which streaks are seen on a vessel it is frequently impossible to decide between a syphilitic, an albuminuric, and a senile sclerosis. As pathological anatomy is bringing atheroma into closer relationship with syphilis, and as arterial sclerosis is gaining a greater relationship with albuminuria, it is difficult to obtain the undoubted truth in any given case. This is further true because the investigation of diseases of the vessels becomes indispensable to utilize only such cases as can be clinically and eventually anatomically proved as uncomplicated as possible. It is a well-known fact that it is in those cases of

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[1] Zeitschrift für klinische Medicin, 1889, Band xvi.

contracted kidney that are especially prone to induce disease in the vessels of the eye that albumin, if present at all, exists in the urine in minute quantities. It is also of special importance that thorough search be made for the possibility of the existence of syphilis.

In the writer's experience, the resulting white changes in the vessels in angio-sclerosis are not of frequent occurrence in simple senile sclerosis. If they are observed, the suspicion of the presence of albuminuria or of syphilis must arise. The arteries seem to display the disease of the vessel-walls more frequently than the veins do. Phlebo-sclerosis appears frequently in the form of wall-thickening, which is invisible ophthalmoscopically. More than once has the writer observed secondary opacity of the walls appear in a branch of the retinal artery in senile patients, this appearing some time after obstruction of its accompanying vein, or as a result of so-called branch-thrombosis of the central vein. Opacity, not only in the true wall of the arteries and veins, but also in the adventitia, is seen in the retina. The writer has noticed this more frequently in the veins than in the arteries. He has found that it is more pronounced in cases of obscure disease of the vessels, with numerous hemorrhagic extravasation, that occur in middle age, these probably being of the nature of hereditary syphilis. Frequently it is impossible to determine how much of the lumen of the vessel is constricted when opacity of its walls exists. The permeability of an apparently completely degenerated vessel can often be determined by the fact that on the side that is situated beyond the opacity the vessel contains blood. In ophthalmoscopically invisible thickening of the wall, on the contrary, it is possible sometimes to judge of the constriction of the lumen of the vessel by the width of the blood-column. Also, often in a single branch there is a localization of the disease which is exactly like that found by Thoma and others in other parts of the body. The constriction of the blood-columns thus produced can give rise to a condition which has been pictured ophthalmoscopically by Raehlmann, Friedenwald,[1] and others, and which at times can be diagnosticated with a reasonable degree of certainty. The irregularity in the calibre of the arteries and veins is seen not rarely in old persons who, owing to hemorrhages in the retina, present to a certain degree the picture of so-called venous branch-thrombosis.

Irregular, knotty-like, or spindle-shaped configuration of the vessel is often aggravated in the vein by the fact that not only constrictions but dilatations are present, these being often so marked that they might be spoken of as varicose degenerations. Generally, a single spindle or sacculate—a mere localized relaxation of the vessel-walls, as the result of circumscribed sclerosis—may be present. Whatever may be the significance of the new vessel-formation, it is well recognized to be a product of inflammation. It occurs principally within the anterior portion of the retina, and under some circumstances it can be detected microscopically. There can be no doubt

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[1] Archiv für Ophthalmologie, Band xxxiv.

that aneurism, as well as varicose dilatation of the retinal vessels, readily gives rise to hemorrhages. Narrowing of the calibre of the vessel from proliferation of the intima causing stagnation of the blood has the same tendency. In consequence of the fact that the more marked the degeneration of the wall becomes the quicker the extravasation will occur makes the appearance of hemorrhage the most important symptom in angio-sclerosis. Hemorrhages under the conjunctiva, in the retina, in the vitreous chamber, and rarely between the vitreous humor and the retina, or between the retina and the chorioid, are frequently the results of senile angio-sclerosis. The more carefully patients are examined for albuminuria and syphilis the more rarely do cases of genuine senile retinal hemorrhages become. In sixty thousand cases seen in the practice of the writer, Ammann[1] found but twenty-two in which retinal hemorrhage appeared as a complication of simple senile sclerosis; and in only a portion of these was the form of degeneration that is associated with white bands or complete white coloration of the vessels seen. In eleven the hemorrhage occurred in the macula lutea alone, or in the conjunctiva in association with retinal hemorrhages. Frequently they simulate the picture of senile macular disease. The writer[2] has called attention to the fact that this important retinal affection is spoken of as being the result of senile vessel degeneration. In the remaining non-macular type of retinal hemorrhages dilatation of this kind is present. Cases have been described in which there is a dilatation of the veins, giving them the appearance of a string of pearls (Liebreich).

That local constriction of branches of the veins where they are crossed by arteries occasionally presents itself ophthalmoscopically, as described by Raehlmann, can be confirmed by the writer. The effect of one vessel upon another at such a crossing point shows itself in a marked proliferation of the intima. An increased tortuosity of the arteries occurs which Raehlmann considers to be of great importance, presuming that it corresponds with the first stage of arterio-sclerosis. In many cases it is easier, however, to see the marked tortuosity which takes place in single venous branches. It is often so pronounced that there can be no doubt of its pathological nature. If it arises from an abnormally distended venous branch, in consequence of obstruction to a neighboring branch, it forms a collateral circulation.[3]

According to Raehlmann and Osten-Sachen, a pulse appears in the veins in angio-sclerosis more frequently than it does in the arteries. This pulsation manifests itself in fluctuations in the calibre of the vessels from the centre towards the periphery, but it can still be seen in the smallest venous branches.

Just as phlebo-sclerosis may produce varicose formations, so does arterio-

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[1] Beiträge zur Augenheilkunde, Heft 33.

[2] International Ophthalmological Congress at Heidelberg, 1888, and Atlas of Ophthalmoscopy.

[3] Klinische Monatsblätter für Augenheilkunde, February, 1898.

sclerosis play an important part in the causation of aneurisms of the retina. Liouville[1] has demonstrated in three aged persons suffering from atheroma miliary aneurisms in the brain. These observations have been confirmed by Sous,[2] Uhthoff,[3] Schmall,[4] Rachlmann,[5] Rampoldi,[6] Mackenzie,[7] Litten,[8] Gowers,[9] and others. The assumption of Thoma[10] that the arterio-sclerotic form of aneurism is the most frequent finds its confirmation in the reports of these cases.

The mode of the development of aneurism in the small vessels of the retina in arterio-sclerosis requires further investigation.

On the average, the patients were over sixty years of age, and were subjects who had performed hard manual labor throughout their lifetimes.

According to the findings of Ammann, aside from the cases of macular hemorrhage, the prognosis for sight is not hopeless.

The hemorrhages in so-called thrombosis of the central vein or its branches can, in the majority of instances, be attributed to a senile form of arterio-sclerosis, as in a large portion of them obliteration of the veins exists as the result of a phlebo-sclerosis. As a result of the anatomical studies of Schnetterman, the writer is convinced that this process leads in places to a constriction of the veins, so that in many instances "angio-sclerosis obliterans" may be spoken of.

The writer is of the opinion that angio-sclerosis can, on the one hand, through a gradual narrowing of the arteries, produce a picture that is similar to embolism of the central artery, and, on the contrary, through a narrowing of the veins, evidence a disturbance which coincides with thrombosis. In chorioiditis-gyrata, which Fuchs has recently described, the writer has seen this sclerosis well developed. Finally, arterio-sclerosis may bring about quite serious disturbance of the eye through the optic nerve at its entrance into the skull-cavity,—becoming compressed by atheroma of the ophthalmic or carotid arteries, with the result of producing a partial or a complete atrophy, as pointed out by Ware,[11] Mackenzie,[12] Bernheimer,[13] and Sachs and Otto.[14] This pressure-atrophy of the optic nerve would be even more pronounced in cases in which aneurismal dilatations of the vessels at the base of the brain were affected, as have been reported in cases seen by Mac-

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[1] Gazette des hôpitaux, 1890.
[2] Annales d'Oculistiques, 1855.
[3] Bericht der ophthalmologischen Gesellschaft zu Heidelberg.
[4] Archiv für Ophthalmologie, Band xxxiv.
[5] Zeitschrift für klinische Medicin, 1889.
[6] Annali di Ottalmologia, 1889.
[7] Royal London Ophthalmic Hospital Reports, ii.
[8] Berliner klinische Wochenschrift, 1881.
[9] Ophthalmologie.
[10] Archiv für Ophthalmologie, Band xxxv., 1889.
[11] Observations on Cataract, vol. ii.
[12] A Practical Treatise on the Diseases of the Eye.
[13] Verhandlung des internationalen medicinischen Congresses in Berlin, 1890, Band iv.
[14] Untersuchungen über Sehnerveränderungen bei Arterio-Sclerosis, Berlin, 1893.

kenzie, Michel,[1] and Nothnagel.[2] Lurje[3] has detailed the examination of thirty eyes from old patients suffering from arterio-sclerosis in which the basal cerebral arteries also exhibited sclerosis.

## ALBUMINURIC ANGIO-SCLEROSIS.

Although a great deal of study,[4] both microscopically and ophthalmoscopically, has been devoted to the vessel-degeneration which occurs in chronic albuminuria, still many gaps remain, especially in the histological knowledge. On the whole, the changes are similar to those which occur in senile angio-sclerosis, only that they are frequently much more developed, so that the vessels,—from the opacity of their walls,—particularly in their ramifications, at times reveal the disease (Mules,[5] Hulke,[6] de Wecker,[7] Gowers,[8] Harlan[9]).

Whether the process originates in the intima, which is most likely, or in the media, and how greatly the adventitia is involved, and whether a periarteritis or a periphlebitis plays any *rôle*, requires further investigation. The process principally affects such vessels as are especially prone to invasion by obliterating endarteritis, which is also found in the kidneys, and as a result of which—through a gradual increasing thickness of the walls of the vessels—these organs gradually contract. The original proliferation of the intima gives place later to a hyaline and a fibrous degeneration of the vessel-walls, which may become quite homogeneous. In many cases, however, fatty degeneration, especially of the adventitia, supervenes in the form of the so-called "fatty granule cell."[10] In diabetes, instances have been described that have been associated with sclerosis of the retinal arteries, and with hemorrhages and degenerative areas of the retina. That the retinal changes do not necessarily arise from albuminuria is shown by the cases of Desmarres,[11] Noyes,[12] and Haltenhoff.[13] Retinal hemorrhages have been observed in polyuria by Galezowski. In a case observed by the writer, recovery took place notwithstanding a long-continued, severe, retinal hemorrhage.

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[1] Archiv für Ophthalmologie, Band xxiii.

[2] Wiener medizinische Presse, 1884.

[3] Dissertation, Dorpat, 1893.

[4] Handbuch der gesammten Augenheilkunde; Transactions of the Ophthalmological Society of the United Kingdom, vol. i.

[5] Transactions of the Ophthalmological Society of the United Kingdom, vol. ii., 1882.

[6] Royal London Ophthalmic Hospital Reports, vol. vi.

[7] Traité des Maladies des Yeux, ii. 316.

[8] Medical Ophthalmology, p. 835.

[9] Transactions of the American Ophthalmological Society, vol. iv.

[10] Klinische Monatsblätter für Augenheilkunde, 1889.

[11] Traité des Maladies des Yeux, 1858.

[12] Transactions of the American Ophthalmological Society, 1869.

[13] Klinische Monatsblätter für Augenheilkunde, 1878.

## SYPHILITIC DISEASES OF THE VESSELS.

Diseases of this type claim a special consideration, for the reason that they can present the same appearances as senile and albuminuric degenerations, and also because such authorities as Michel and Alexander closely associate syphilitic diseases of the interior of the eye with specific arteritis. That the vessels of the iris are markedly involved in syphilitic inflammation was first recognized by Michel and Fuchs. Uhthoff and Bach found the same condition in the retina. Besides these greater inflammatory changes of the arteries, there is still another syphilitic form of disease of the vessels which greatly resembles that seen in ordinary senile sclerosis; in consequence of which it is difficult to assert whether the process is more or less inflammatory in nature. This is "endarteritis specificans." At the present time, the prevailing opinion is that syphilitic diseases of the vessels are so irregular in their nature and manifestations that their existence very frequently can only be deduced by the presence of concomitant syphilitic lesions. Baumgarten considers the tendency to a formation of small nodular syphilomata in the adventitia as characteristic of syphilis, but, according to others working in the same line, ophthalmologists are apt to assert a diagnosis of syphilitic disease of the eye before any actual anatomical proof is presented. Further investigations are necessary to determine whether there are inflammatory and non-inflammatory processes in this affection, as suggested by Michel, Fuchs, and Bach. Such a division would appeal to the physician for its advantage in a clinical study of these conditions. Syphilis not only induces inflammatory changes, especially in the retina, but occasionally produces conditions that are similar to those that are seen in senile or albuminuric sclerosis. Blood extravasations, opacities of the vessels, and occasionally retinal opacities are seen. Hirschberg reports a case in which periarteritis occurred in the retina, exciting symptoms similar to endarteritis, but of a more inflammatory type. Syphilitic endarteritis is a rare disease. The writer has seen but nine instances, and Ammon found in his clinic of former times only three unmistakable cases that belonged to this class. The disease-picture is composed of the following varying component lesions:

1. Visible opacity of the walls of the arteries and rarely of the veins, so that the blood-columns of the diseased vessels are accompanied with lateral white lines; or the blood-columns or the vessel-walls may be covered in places with a grayish-white opacity. At times, complete obliteration of the terminal twigs takes place.

2. As in senile sclerosis, an almost invisible disease of the vessel-walls may be present, only manifesting itself by a narrowing of the blood-columns. As a result of both of these types, extravasations of blood from the diseased walls can take place. Consequently, hemorrhage appears, not only in a position at which a white degenerated vessel ramifies (Fig. 8, on

the lower left-hand side), but also within the area of vessels that are apparently unaltered (Fig. 8, on the right side, below).

3. They also may show their changes by an irregular filling of the vessel. Further, a vessel can exhibit a whitening of its walls, but is not accompanied by hemorrhage; or if such blood extravasations appear, they will evidence themselves in the periphery of the vascular branches.

4. Under some conditions, an opacity corresponding to that occurring in an obstruction of the central artery or one of its branches may be seen, this appearing as a gray-white or a milky area with ill-defined edges. While, however, in the obstruction of an arterial branch, generally no hemorrhages, or only slight ones, occur in the area of opacity, yet in this condition considerable hemorrhage may take place within the opaque retinal tissue. This opacity can be so extensive that it may closely resemble detachment of the retina. The circumpapillary portion of the retina is often slightly hazy and of a grayish tint.

5. Groups of circumscribed white splotches, similar to the degenerative splotches seen in albuminuria, more rarely occur.

In one-half of the writer's cases, the macula was markedly affected; four times by hemorrhage and once by fine pigmentation, as is seen in hereditary syphilis.[1]

Slight optic neuritis is a further accompaniment which has often been described. Traces of iritis can of course be present. Very frequently the other eye will show traces of some form of syphilitic disease.

The subjective symptoms vary greatly, according to the location of the principal changes in the retina; the rule being that the more severely the macula suffers the poorer will be the vision. The disease generally belongs to the later stages of syphilis. The subsequent course of the disease shows great tendency to recidivity, especially when the treatment cannot be energetic and prolonged.

In syphilitic disease of the retinal vessels, the writer has so frequently seen the opacity of the walls appear as fine, disseminated scales upon the arteries, that he attaches considerable importance to this condition. Other similar vessel-degenerations rarely show this appearance. As a rule, these small scale-like flakes are only perceptible when the examination is made with the upright image. In many cases, a characteristic appearance is formed by the milky opacity of the retina in the region of the diseased vessel. Frequently, the manner in which the disease is affected by treatment gives the first clue to the diagnosis. Difficulty may be experienced in the significance of the retinal strings, such as figured by Jäger and those seen in the writer's Atlas. They are never found to be continued as vessels, and they often have courses that are impossible for vessels,—*e.g.*, transversely across the retinal vessels. If the vitreous humor is hazy, the differentia-

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[1] Atlas der Ophthalmoskopie, Taf. 81.

Fig. 8.

Fig. 9.

Reprinted from the Atlas of Ophthalmoscopy by O. Haab.

Lith. Anstalt F. Reichhold. München.

tion becomes difficult. The more inflammatory conditions of perivasculitis may produce similar pictures in the retina.

The tendency to relapse must be borne in mind, although the disease, if properly treated, may terminate favorably. The writer has seen marked disease of the macula which has produced a reduction in vision to one-twentieth of normal so improve that sight has risen to two-thirds. The treatment, however, must be energetic and long continued.

The writer has never seen the chorio-retinitis noted by Oswalt and Hirschberg, although he has searched for it for ten years in all syphilitics. According to the former author, this affection exhibits itself by the presence of small, round, bright spots, that are similar in appearance to a bunch of grapes, these being situated not only in the periphery of the eyeground but also in the macular région.

## NEW VESSEL-FORMATION.

In senile sclerosis, new vessel-formation, as a rule, assumes only moderate proportions, while in syphilis, it is more frequently present. Often the process begins with hemorrhage into the retina or into the vitreous humor, and the vessels are not seen until the blood is so far absorbed that a view of the fundus becomes possible. They appear to form either small loops or large net-works extending into the vitreous chamber. Usually, the optic nerve or its surroundings is the point of origin for this vascular growth. As this formation is frequently associated with hemorrhage, it is believed to be related to the so-called "retinitis proliferans," in which the connective-tissue new-formation upon the inner surface of the retina proceeds, as a rule, from hemorrhagic extravasations. This vessel-growth takes place more frequently after hemorrhages of syphilitic origin than after those that are due to albuminuria, senility, diabetes, etc. Probably hereditary syphilis may also be a cause of this vessel growth.

Eyes which have been enucleated for hemorrhagic glaucoma exhibit, as a rule, disease of the vessels to a pronounced degree. The writer can confirm the statements of Edmunds and Brailey, Panas, Wagenmann, etc., who have given this matter careful study. In juvenile eyes with glaucoma, in which senile sclerosis cannot be taken into consideration, hemorrhages occasionally occur, suggesting the belief that arterio-sclerosis stands in etiological relationship with glaucoma.

## RELAPSING HEMORRHAGES IN YOUNG PERSONS.

This apparently causeless type of hemorrhagic affection was known to A. von Graefe, who ascribed it to disease of the vessels. Recent observations, including those of the writer, seem to justify this classification. Notwithstanding the studies of Hutchinson, Nieden, Panas, Chodin, Kipp, and others, the causative factor is still in the dark. From twelve cases at the disposal of the writer, Ammann has noted that in the majority of these the hemorrhages into the vitreous humor were accompanied by changes in

the retinal vessels. What appears to the writer to be of more importance is the fact that in most of these observations, where bright-bordered vessels were to be seen, white degenerative spots could be observed in the retina. At first, these formed a stellar arrangement like that of albuminuric retinitis in the macular region. (Fig. 9.) They then left behind only small, bright, yellowish-red areas in the macular region that were alone visible with the upright image.

The retinal changes can be plainly seen in Fig. 9, in which a small vein coursing in and up has white borders. In this case, it is worthy of mention that the patient—a twenty-five-year-old subject—lost the sight of the fellow-eye through hemorrhagic glaucoma five years previously. The type of the hemorrhage was also of interest. It lay between the vitreous humor and the retina and was semicircular in form. When the head was inclined, the fluid blood always assumed the horizontal position. This change of form is not characteristic of this hemorrhagic process, as it is always present when the upper margin of a hemorrhage lies between the vitreous and the retina. As long as the blood remains fluid it possesses this characteristic movable upper border. The writer agrees with Holmes Spicer and Dimmer when they assume that this form of hemorrhage takes place rather slowly from a retinal vessel,—that is, from a vein. On the contrary, it is to be observed that relapsing hemorrhages of the young do not exhibit a great tendency to lie between the vitreous and the retina, but that they occur more frequently as a single hemorrhage, which may, of course, affect young people from a cause as little understood as is the relapsing form of hemorrhage.

The relatively good prognosis of these half-moon-shaped hemorrhages between the vitreous and the retina is evidenced in the patient whose eye-ground is shown in Fig. 9. Notwithstanding frequent relapses which occasional examination showed to have taken place during three years' time, satisfactory recovery took place. According to the observations of the writer, the number of relapses in general is not the only factor in the prognosis, and does not influence the question of cure as greatly as one might imagine. In the writer's cases, the hemorrhages were repeated from one to three times. In one case they recurred five times and once seven times, and yet the termination in this latter case was favorable. In the writer's experience large vitreous hemorrhages are particularly serious, and when both eyes are not affected from the outset, only the one eye is attacked. Nieden in three cases saw the hemorrhages in two eyes, but never found them affecting the two simultaneously.

Unfortunately, the writer's cases do not assist in clearing up the question of etiology. Schieffel describes a case in which the first manifestation of hereditary syphilis was a high-grade change in the veins of both retinas in the form of a perivasculitis, with which was associated a prolonged hemorrhagic process in the retina without vitreous extravasation. Healing progressed under the use of inunctions. In a case seen by Panas, which

terminated in bilateral blindness, the patient was afflicted with enormous hypertrophy of the heart. Goutard reports two such cases. Frequently recurring nose-bleed has been noticed in many of these instances. The opinion of von Graefe that hemorrhage into the retina comes, as a rule, from the chorioid must be modified. Hemorrhage generally occurs from the retinal vessels and, perhaps, from those that are situated in the extreme periphery. It is well known that the retinal vessels are more predisposed to spontaneous hemorrhage than the chorioidal vessels are. Eales and Nieden believe that the menstruation of the female is a sort of protection against ocular hemorrhages, so that they are rare in women. In the cases seen in women by the writer, the menstruation was disturbed, but, according to this theory, these hemorrhages should not occur in young males who suffer from nose-bleed. It is more likely that nervous and vaso-motor causes are only secondary favoring factors, and that the fundamental disease in very many of these cases is an affection of the vessels of an unknown nature, which causes an abnormal brittleness of the vessels of the eye as well as of those of the nose and skin, which may disappear after a time. Hereditary syphilis probably plays an important *rôle* in the production of the disease.

Young men and more uncommonly young women, between the ages of fifteen and thirty years, are more exposed to the attacks of relapsing hemorrhage. While in good physical condition these patients experience suddenly or gradually a darkening of vision before one or both eyes which is found to be due to hemorrhages in the vitreous. These precede or follow retinal hemorrhages. The absorption of these extravasations occurs quite rapidly, but new hemorrhages soon follow, often more severe, without apparent cause, and frequently at night. These again clear up, and recovery seems to have taken place, when sudden bleedings again take place, until, finally, without visible cause, the process ceases and the eye becomes normal, or nearly so, with the formation of connective tissue either in front of the retina or in the vitreous chamber as a sequel of the disease. In unfavorable cases, the vitreous humor becomes more hazy, resulting in blindness, degeneration of the vitreous body, and retinal detachment. As a rule, inflammatory symptoms are absent, but glaucoma may supervene.

It is well recognized that a single hemorrhagic extravasation may occur in a young subject without any previous history of inflammatory condition in the retina or vitreous humor, which after some time may disappear without leaving a trace of the condition. Anæmia and pregnancy both play certain parts in the production of such types of hemorrhages.

Prognosis in these cases is always doubtful and treatment on the whole is of little value. Pronounced hemorrhages in the posterior part of the vitreous humor and in the retina appear to be less serious than those that are very dense and penetrate into the anterior part of the retina. As a rule, in cases in which hereditary syphilis is the cause, the prognosis is more favorable; but there are exceptions to this statement. Therapy is not entirely powerless. Rest of the eyes and judicious physical restraint

are of the utmost importance. The latter is most useful, as any resulting congestion of the head with increased activity of the heart tends towards rupture of the vessels. In the further treatment—with bodily rest—must be associated a general strengthening. At the same time the employment of mercury and iodide of potassium is to be recommended. In a case in which the process was malignant, the writer would not hesitate to advise ligation of the carotid artery, as has been performed by Mayweg with good results. The employment of periodic venesection to bring about a favorable result has still to be tried.

### THE TOXIC VESSEL-DISEASES OF THE EYE.

That certain poisons circulating in the blood have a deleterious action upon the vessel-walls is apparent to every one. Marked fall of blood-pressure, from whatever cause, is perhaps as dangerous to the vessels and their intima as toxic conditions of the vessel-contents are. Such a fall may also give rise to proliferation of the intima with further changes in the coats of the vessels. Certain poisons may also act in this indirect way by reducing blood-pressure. Other more chronic toxæmias can still further damage the vessels by disturbing the general nutrition, so that the marked anæmia and cachexia that are produced may in themselves lead to degeneration of the blood-vessels.

If the series of poisons that lead to vessel-changes are examined, it will be found:

*A*. Poisons which are introduced into the organism from without and act acutely or chronically. They may be of the nature of alcohol or quinine, or they may be composed of micro-organisms, such as are found in sepsis, malaria, etc.

*B*. Poisons that are generated by the organism itself, such as are seen in uræmia, diabetes, etc. To this division, perhaps, belong the still obscure diseases of the blood and vessels found in anæmia, leukæmia, purpura, etc.

### INTOXICATION-AMBLYOPIA FROM TOBACCO AND ALCOHOL.

If it be assumed that hemorrhages and spindle or nodular forms of the blood-columns are to be accepted as evidences of disease of the arteries and veins, vessel-lesion can undoubtedly be demonstrated in many cases of these types. Ammann (*loco citato*) has found in the clinical history of the writer's cases seven instances in which, together with a more or less well-defined evidence of intoxication-amblyopia, small retinal hemorrhages could be seen situated principally about the papilla and usually overlying a vessel. Recently, the writer has seen other examples of such hemorrhages, and besides (but not in senile patients), has found spindle-shaped irregularities in the columns of blood in the arteries and the veins, which pointed almost certainly to a microscopic thickening of the vessel-walls. Friedenwald and Crawford report a case of spontaneous hemorrhage into the orbit with exophthalmus in which disease of the vessel-walls due to alcoholic and tobacco-poisoning was present.

### QUININE-POISONING.

It will require further investigations to determine whether quinine acts directly or indirectly by diminishing the blood-pressure. In either instance, intra-ocular pressure would aid in diminishing the calibre of the vessels. As far as the vascular lesions go, de Schweinitz has experimentally found an endovasculitis in dogs that were poisoned with quinine. He concludes, however, that quinine acts upon the vaso-motor centres, producing contraction of the blood-vessels with consequent vessel-changes, to which may be added endovasculitis thrombi, terminating in atrophy of the optic nerve (*vide supra*).

### PHOSPHORUS-POISONING.

In this condition, hemorrhages may appear quite early in the retina, even before the vessels evidence fatty degeneration, which later becomes plain in the capillaries and arteries. White degenerative areas similar to those seen in albuminuria are prone to appear. As carbonic-acid poisoning produces fatty degeneration, especially in the arteries, with numerous capillary apoplexies, it is presumable that these changes also take place in the retina.

### LEAD-POISONING.

Oeller has confirmed the belief that lead-poisoning can act injuriously on the blood-vessels. Retinal hemorrhages, endarteritic and perivascular changes in the retinal vessels have all been observed by such authors as Hirschberg, Parisatti, and Melotti.

### SEPTICÆMIA.

Of all the toxic products that are carried into the body by micro-organisms to injure the vascular system of the eye, those of septicæmia are the most important, especially when the disease runs a chronic course, by which anæmia in a greater or less degree is developed. It is recognized, however, that there are cases of general sepsis of known or unknown source (cryptogenetic in type) in which the picture resembles that of pernicious anæmia, even to the retinal findings.

As is well known, two forms of disease arising from the infection of the organism, and especially of the blood by micro-organisms, are recognized: 1, The metastatic or pyæmic; 2, the so-called septic. From a pathologico-anatomical stand-point this division is perhaps open to criticism; for the present, however, it may be employed. In the eye itself also a metastatic process can be separated from a so-called septic one. Besides the malignant metastatic ophthalmia, which leads to rapid purulent inflammation of the eye, it has been possible (through Roth, as the result of anatomical investigation) to recognize a second form of retinal affection that is relatively benign, inasmuch as it is confined to the retina. In purulent infection of the retina disturbances in the vitreous and the chorioid do not appear, and in many of these cases death does not occur. The changes in the retina

consist merely in hemorrhages and white splotches that are quite similar to those that are seen in albuminuria, diabetes, and pernicious anæmia. As the result of the study of nine cases of sepsis, Litten has proved that the retinal extravasations are only a part of a hemorrhagic process which gives rise to hemorrhages on the inner surface of the dura mater and on serous surfaces, such as the pelvis of the kidney, etc. In the retina, the hemorrhages are usually bilateral in character, and in a portion of them, a quite distinct white centre can be seen; this also being the case in many of the hemorrhages that occur in the skin and the pelvis of the kidney. The retinal hemorrhages always appear shortly before death.

Litten and Roth have never found bacteria in septic retinitis. Köhler has seen compound granule-cells and swollen spindle-shaped elements without bacteria as a substratum of the bright speck, in the midst of the retinal hemorrhages. In one case bacterial emboli were found, these, however, being especially noticeable in a capillary which afterwards showed sacculated protuberances and enormous cylindrical dilatation.

While the investigations of Axenfeld show that, by the invasion of micro-organisms, the retinal vessels are the ones that are primarily affected, particularly in cases in which the condition is bilateral, still there is much to be said in support of the view that only the toxines that circulate in the blood in sepsis are capable of injuring the vessels and producing hemorrhages as well as white splotches on the retina. As these retinal hemorrhages are seen in contamination of the blood by inorganic poisons and excretory products of the body, "retinitis septica" may be considered in connection with these systemic poisons.

While, perhaps, in many instances, Weil's disease is the result of sepsis, it is of interest that Herrnheiser has described two such cases in which hemorrhages into the retina formed a remarkable feature of the disease. Very likely the retinal hemorrhages that have been observed by Thoma, Knies, and others in extensive burns of the skin arise from sepsis. It might be assumed that a septicæmia results from the absorption of a great amount of decomposed products from the burned skin. A tendency to fatty degeneration and hemorrhage is found in all the tissues. Hyaline thrombotic formations of blood-plates (Welti) are also seen. Retinal hemorrhages are of serious prognostic import, but they are not of absolutely fatal significance.

In malaria, the diseased conditions of the eye-ground are in part of an inflammatory nature (neuritis, infiltration of the vitreous humor, and chorio-retinitis), and in a measure are similar to those that are seen when the blood possesses toxic qualities,—*i.e.*, hemorrhages with a bright centre and white splotches. Von Kreis has observed hemorrhages in the retina and the vitreous humor. Despagnet has seen marked constriction of the retinal vessels which he considered to be due to an arteritis, whereas Ramorius regarded the condition as dependent on spasm.

If the following group of diseases is assumed to be caused by auto-intoxication, the hypothesis will be more significant than though they were

considered as resulting from constitutional anomalies that mean but little. The least that can be asserted is that the changes are the result of the presence of materials in the blood that operate as ectogenic poisons, this being so because the retinal changes that are produced by these poisons appear alike; while, in addition, the pathological appearances in the retina strengthen the belief that in an intense anæmia, leukæmia, etc., toxines in greater part create the symptoms that are produced in the body.

In anæmia and chlorosis, the ocular conditions, aside from any asthenopic symptoms, are unimportant. Blanching of the papilla, underfilling of the blood-vessels, and pallor of the blood-columns are the only signs that are present in well-marked cases; and then not always. In chlorosis, an arterial pulse which manifested itself as a fluctuation of the vessel calibre and a visible locomotion of the blood-current is more frequently present. In this disease, optic neuritis, which rapidly subsides under the use of iron, has been repeatedly observed. In a case of high-grade chlorosis, without other disease, Knies once saw brilliant white spots appear in the retina; these being arranged principally in the form of the macular star seen in albuminuric retinitis. Vision, however, was but little affected. The rest of the eye-ground was normal. The affection was monocular, lasting for more than six months, and disappeared without leaving a trace.

In pernicious anæmia, the changes are more pronounced. Aside from degeneration of the external eye-muscles, which Fränkel describes as pale clay-colored and devoid of transverse strata, hemorrhages in the retina are met with which are greater than those that are seen in anæmia; these being invariably present in cases in which death results. Sometimes in the fatal cases, white spots are also seen, so that the picture is exactly like that of albuminuria, diabetes, and septicæmia. (*Vide* Fig. 2, facing page 504.) The cases in which hemorrhages are alone present are more frequent, these possessing the peculiarity of having white centres, to which Quincke has referred. This white centre is also seen in the hemorrhages of septicæmia and in other retinal extravasations. In the opinion of the writer, it occurs more frequently in hemorrhages that are associated with toxic factors.

These retinal hemorrhages are sometimes only present in the region of the optic nerve. In such cases they are lengthened out and radiating. At other times, they are distributed throughout the fundus, ranging in size from the diameter of the principal branch of the retinal artery to that of the papilla itself. Occasionally, it will be noticed that the entire eye-ground is somewhat pallid. It must always be remembered, however, that this symptom is not always to be relied upon, as the color of the eye-ground is more greatly dependent on the quantity of pigment than it is on the color of the blood. The subjective symptoms are sometimes slight, but when the vision is carefully tested it will often be found to be greatly reduced. Sight is, of course, most endangered when the macula is affected, which, on the whole, however, occurs quite infrequently.

Œdematous swelling of the papilla may be present. The bright

centre of the hemorrhage is due not to accumulation of leucocytes but to heaping varicosed nerve-fibres, as in septic hemorrhages (Uhthoff and de Schweinitz). Further, Uhthoff found deposits of colloid and fine granular masses in the inner nuclear layer. As yet, no important changes have been determined, notwithstanding that not rarely the hemorrhages have been seen about the vessels. (Fig. 7, *vide* vein.) In the chorioid, the vessels are found to be packed with red blood-vessels, which explains further why pallor of the eye-ground cannot be often discerned, even in fatal cases. This marked filling of the chorioidal vessels was observed in the case which is pictured in Fig. 2. In this case, vessel-changes in the retina could not be seen in the retina. In contrast, Ammann has seen in another case, although in quite a limited area, an endarteritis obliterans in a peripheral artery of the retina, the patient being but forty-eight years of age. These retinal changes are found in various cases of severe anæmia, thus, for example, not only in pernicious anæmia terminating in death, but also in patients who convalesce from this disease. Complete disappearance may occur in these cases, as the writer has seen take place in a surprising manner in a case which is reproduced in Fig. 49 of his Atlas. He has often observed retinal hemorrhage and white spots in subjects who were impoverished in blood; for example, by carcinoma of the stomach. An especially interesting form of anæmia occurs in connection with helminthiasis, particularly in that form which is produced by the bothriocephalus. This variety of anæmia has acquired a considerable importance also in the explanation of pernicious anæmia, as it has been shown that in many cases, especially in those in which the worm remains in a dead state in the intestines, a pernicious form of anæmia may be excited.

Anæmia arising from ankylostoma can lead to retinal hemorrhages. In reference to the etiology of pernicious anæmia it may be noted that a similar disease occurs in horses. Akin to pernicious anæmia are the diseases that are especially characterized by acquired hemorrhagic diathesis; such as scurvy and purpura. The view has often been expressed that infectious processes are active, especially in the form of scurvy, in which conjunctival hemorrhages may occur and extravasations into the retina take place.

In purpura, hemorrhages are more frequently found in the retina than they are in scurvy. Gowers believes that they are always present in severe cases. They are usually striated in form, and appear most numerously in the vicinity of the papilla. White spots in the retina with a marked blurring of the edge of the papilla have also been met with.

In leukæmia, the hemorrhagic diathesis manifests itself by phenomena that are, as a rule, similar to those that are seen in the above-described diseases. Frequently, nothing is found in the retina, or, at most, a somewhat dilated vein, whose color is paler than normal. The picture of "leukæmic retinitis" found in Liebreich's Atlas will, as a rule, be looked for in vain. The light color which he gives the fundus is occasioned principally by the fact that there is but little pigment. In the experience of

the writer, hemorrhage in leukæmia appears much earlier than in pernicious anæmia, especially when the blood contains fifty per cent. of blood-corpuscles. As a rule, these hemorrhages lie in the nerve-fibre layer.

Besides the hemorrhages, areas of bright degeneration are seen mostly occupying the periphery or the macular region. They are composed of leucocytes. What has impressed the writer is the bright color of the medium-sized and, especially, the appearance of the finer vessels, which have the appearance as though they were sclerosed. Diffuse opacity of the retina, particularly around the papilla, but situated farther out, is often observed.

Anatomically, manifold conditions are present in the substrata of the white areas. Nerve-fibres, fat granule-cells, and extravasated leukæmic blood which contains a large number of white corpuscles, are all seen. Hemorrhages are also found in the chorioid. The retinal opacity is explained by a hypertrophy of the retinal connective tissue.

Hæmophilia, a congenital hemorrhagic diathesis, occupies a peculiar position. At present it is but little understood, especially the acquired form. Spontaneous and post-traumatic hemorrhages into the orbit and retinal hemorrhage in the form of retinitis proliferans have all been observed.

# EYE-DISEASES AND EYE-SYMPTOMS IN THEIR RELATION TO ORGANIC DISEASES OF THE BRAIN AND SPINAL CORD.

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THE organic diseases of the brain are either focal or diffuse.

## FOCAL DISEASES OF THE BRAIN.

In respect of their symptoms, focal brain-diseases may be conveniently classed under two heads,—namely, 1, those which cause an increase of the intra-cranial pressure, and, 2, those which have not that effect.

The focal brain-diseases which produce an increase in the intra-cranial pressure are tumors, including syphilitic and tubercular growths, aneurisms, parasitic and simple cysts, and even abscesses.

The focal brain-diseases which are unaccompanied by increased intra-cranial pressure are chiefly hemorrhages and softenings.

Cerebral tumors, by reason of the increased intra-cranial pressure which they cause, are accompanied by certain symptoms which do not belong to other brain-diseases. These are termed general or diffuse symptoms, and indicate the presence, but nothing more, of an intra-cranial tumor. Of these symptoms, the three principal are double optic neuritis, headache, and vomiting or nausea; and there are often also vertigo, drowsiness, mental lethargy, and attacks of temporary total loss of sight.

In cerebral hemorrhage there is not the continuous high pressure usual with cerebral tumor, or else, should the hemorrhage be copious enough to cause increased intra-cranial pressure, it produces a fatal result before optic neuritis can come on.[1]

Tumors, moreover, and also those focal cerebral diseases which do not cause increased intra-cranial pressure, are, as a rule, attended by what are known as direct, focal, or localizing symptoms,—symptoms, that is, which indicate the seat of the lesion, under whichever of the above two great

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[1] In some rare cases of extravasation of blood at the base of the brain, double optic neuritis is seen (pp. 582 and 620); but there the *modus operandi* is different from the one under consideration.

heads it comes. These localizing symptoms are either paralytic or irritative, according as the part affected is destroyed or irritated by the lesion; and they enable an opinion to be formed as to the part of the brain in which the lesion is situated.

Finally, we have to recognize a class of symptoms which the writer calls distant symptoms, but which are more commonly known as indirect symptoms, as pressure symptoms, and as pseudo-localizing symptoms.[1]

These distant symptoms are not the result of the local disorganization caused by the lesion, but, it may be, of its pressure, of disturbances of circulation, of inhibition, or of reflex irritation, interfering with the functions of organically healthy parts of the brain more or less distant from the lesion. Distant symptoms are sometimes very ephemeral; but when they last long they are a fertile source of confusion to the clinicist, simulating direct symptoms as they then do. Cerebral tumors are much more prone to be attended by distant symptoms than those lesions which cause no increase of brain-pressure.

But the reader must here be reminded of the remarkable fact that focal brain-disease is sometimes latent, in so far as it may give rise to no direct symptoms. There are few parts of the brain in which a lesion has not been found without its having produced any localizing symptoms; and lesions in those parts which, as a rule, give rise to localizing eye-symptoms may in this respect be latent, like lesions elsewhere. Absolute latency of a growing cerebral tumor, if it ever occurs, does so very rarely. (Obernier,[2] Bramwell.[3])

The two eye-symptoms—double optic neuritis and attacks of temporary total loss of sight—which occur as general, or diffuse, symptoms, indicating merely the presence, but not the position, of a tumor of the brain, will now be discussed. The former of these is a symptom of the first importance, while the latter is of much less value. Afterwards the localizing eye-symptoms caused by focal lesions of both kinds, in various parts of the brain, will be described.

## GENERAL, OR DIFFUSE, EYE-SYMPTOMS OF INTRA-CRANIAL TUMOR.

**1. Double Optic Neuritis, or Papillitis.**—It was formerly thought that one special form of papillitis was pathognomonic of intra-cranial tumor,—namely, that in which the swelling of the papilla is great and

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[1] We cannot assume that these symptoms are less the direct result of the lesion than any of the others which are present, and in many instances at least it is certain that they cannot be due to pressure. In short, we do not yet know what produces these symptoms; we know only that they are the result of interference with the functions of parts of the brain not actually involved in the lesion, and at a greater or less distance from the latter, and the term "distant symptom" conveys this idea, although not quite satisfactorily, without committing us to any theory. The corresponding German term is "Fernwirkung."

[2] Ziemssen's Cyclopædia of the Practice of Medicine, xii. 241.

[3] Intra-Cranial Tumors, p. 20.

abrupt and its margin defined, while the central artery is much diminished in calibre and the central vein engorged. This is the form which was termed "Stauungspapille" (congestion papilla, choked disk) by von Graefe, and it is undoubtedly the form of papillitis most suggestive of intra-cranial tumor, and most commonly found with it. But we have come to learn that this same form sometimes occurs under other conditions, and also that a cerebral tumor may be attended by a papillitis in which the swelling is not pronounced nor its margin well defined, and where there is little or no alteration in the calibre of the vessels. In cerebral tumors, moreover, the optic neuritis is sometimes accompanied by retinal changes (hemorrhages and white spots) not unlike those found in albuminuric retinitis, and in Bright's disease, if uræmia should be present, the whole clinical picture may be very like that of a cerebral tumor, hence in all cases of double optic neuritis the urine should be tested. In lead encephalopathy, double optic neuritis is common (p. 627) as also in meningitis (p. 618) and in hydrocephalus (p. 621), and it occurs in some cases of disseminated sclerosis (p. 612) as well as of diffuse sclerosis of the brain (p. 614), in rare cases of hemorrhagic extravasation at the base of the brain (p. 620), and sometimes in anæmia. Cerebral hemorrhage, as such, is rarely, if ever, the cause of optic neuritis. Should these two conditions be diagnosed to coexist, the presumption in favor of the presence of an intra-cranial tumor as the cause of the hemorrhage is strong, plumbism (p. 618) and renal disease having been excluded, and glioma is the form of tumor most likely to be associated with cerebral hemorrhage.

Mauthner laid down the rule that for the diagnosis of typical congestion papilla a prominence of $\frac{2}{3}$ mm. is required ($\frac{1}{3}$ mm. of prominence $= +1$ D). And other authors have adopted this rule. The prominence may attain a height of 2 mm.

Optic neuritis is usually the first or one of the first symptoms of cerebral tumor; but sometimes it does not develop until a later stage, while in about twenty per cent. of the cases it is not present at any period. It is a valuable sign, owing to its usually early appearance, its great frequency, and its objectiveness. In cases of tumor of the frontal lobe a very late appearance of congestion papilla is by no means uncommon. In its initial stage the congestion papilla may not present a typical appearance.

As Byrom Bramwell tersely puts it,[1] "The absence of double optic neuritis does not necessarily exclude the presence of a tumor; but the fact that there is no optic neuritis does suggest doubt; and, unless the other symptoms of tumor are very clearly defined, or unless the physician feels satisfied that there is no condition present except tumor which could reasonably be expected to account for the phenomena of the case, he will be wise, in the absence of double optic neuritis, to hesitate before committing himself to a positive diagnosis."

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[1] Intra-Cranial Tumors, p. 41.

The papillitis does not at first affect the sight, which in most cases remains perfectly normal for a long time; consequently, no diagnostic value attaches here to an examination of the acuteness of vision. The presence or absence of papillitis can be determined only by the ophthalmoscope. Gradually some contraction of the field of vision comes on, and the central vision is lowered. This contraction of the field must not be mistaken for a localizing symptom. Indeed, where neuritis is present, the findings of the perimeter are lessened in value as aids to localization, for we cannot always tell whether a given defect in the field is to be regarded as due to the neuritis or to the position of the destructive lesion in the brain. The former is capable of causing defects in the field which might easily lead the observer astray, even well-defined homonymous hemianopsia having been sometimes produced by it, or such defects may be merely functional in connection with the cerebral disease. As the case advances, the field becomes contracted from all sides, and the perimeter shows an extension of the blind spot beyond its normal dimensions, and occasionally an absolute central scotoma is developed. Ultimately, in nearly all these cases, unless death intervenes or the cranium is trephined, complete blindness ensues, and, should the neuritis subside, optic atrophy sets in. But where the papillitis is caused by a syphilitic gumma of the brain, and an active mercurial treatment is adopted at an early stage, the papillitis very often disappears, leaving a permanently sound optic nerve, with perfectly normal vision.

The papillitis is rarely in one eye only, although it is often somewhat more marked in one than in the other. Some authors state that in monocular optic neuritis due to cerebral tumor the papillitis is usually in the eye which is on the same side as the tumor; but Dr. Hughlings Jackson in two cases[1] saw uniocular neuritis in the eye away from the hemisphere which contained the tumor, so that clearly no conclusion can be drawn from the occurrence.

A tumor anywhere in the brain is capable of producing optic neuritis. It is not required that the tumor should be in or adjacent to some part of the brain concerned in vision. Even small and slowly forming growths may cause papillitis; but, as might be presumed, the large and rapidly growing tumors are those most apt to induce it. The increase in the intra-cranial pressure is not always due to the presence of the tumor merely, but also to dropsy of the ventricles, which is frequently present in these cases to an extreme degree. This ventricular dropsy largely accounts for the occurrence of papillitis with small cerebral tumors, for it is often not less well marked with them than with the large tumors. Some writers state that tumors of the basal ganglia and cerebellum are more constantly associated with optic neuritis than are tumors of the convexity, in consequence, it is thought, of the fact that they oftener produce ventricular dropsy by closing the communication between the ventricles or by pressure

[1] Transactions of the Ophthalmological Society of the United Kingdom, i. 67.

on the venæ Galeni. Indeed, as will be mentioned again later, the acquired hydrocephalus of adult life is capable of causing all the symptoms of cerebral tumor without any tumor being present.

Cerebral cysts do not often cause optic neuritis. It has also been generally held that cerebral abscess is not very commonly a cause of papillitis, but this is not the view held by Macewen, of Glasgow, whose experience in this department is extensive. He states[1] that optic neuritis is frequent in cerebral abscess, especially in the later periods of the disease,—towards the end of the second stage,—and in those abscesses which have attained a considerable size. Cerebral abscess being an acute affection, and papillitis seldom commencing at the outset of the disease, it will not, he says, as a rule, have attained great intensity when first observed; but it is progressive, advances at a rapid rate, and may continue to do so for some time after the evacuation of the abscess. It is rarely followed by neuritic atrophy. It is not always more intense on the side of the lesion, and the eyesight is seldom much affected. Abscesses in the frontal and temporo-sphenoidal lobes cause it, and those in the cerebellum; but it is not found with small abscesses, nor with those that run a very rapid course. Absence of optic neuritis does not exclude cerebral abscess, but when it is present it is a distinct aid to the diagnosis.

Sometimes atrophy, and not neuritis, will be the diseased condition of the optic nerves found with intra-cranial tumor. For the most part this atrophy is of the post-neuritic variety; yet, doubtless, optic atrophy without any sign of preceding inflammation is occasionally seen in cases of cerebral tumors, and is probably the result of direct pressure on the tracts, chiasma, or intra-cranial portion of the optic nerves by the tumor, or by a distended fourth ventricle.

Diseases in which the ocular and other symptoms sometimes resemble those in cases of cerebral tumor are diffuse sclerosis of the brain (p. 614), acquired hydrocephalus of advanced life (p. 621), and encephalopathia saturnina (p. 627).

*Pathogenesis.*—The causation of optic neuritis in cases of cerebral tumor is still a subject for debate. Von Graefe taught that the increased intra-cranial pressure impeded the flow of blood through the cavernous sinus, and, consequently, the return of blood from the central vein of the retina, and that the congestion in the latter vessel was further promoted by the unyielding nature of the sclerotic foramen, and hence the œdema of the papilla. But this purely mechanical theory had in the main to be abandoned when the free anastomoses of the superior ophthalmic vein with radicals of the facial vein were demonstrated.

It was subsequently shown that in many, if not in all, of these cases the intervaginal lymph-space of the optic nerve was distended with lymph which was presumably driven out of the subarachnoid space by the high

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[1] Pyogenic Infective Diseases of the Brain and Spinal Cord, Glasgow, 1893, p. 141.

intra-cranial pressure; and Schmidt-Rimpler[1] and Manz[2] put forward the view that the outflow of the venous blood from the eye was impeded by this dropsy of the sheath, and that the neuritis was due also, in part, to stagnation of the lymph in the papilla itself. Leber[3] and Deutschmann[4] hold that the dropsy of the sheath produces the neuritis not so much by compressing the head of the optic nerve and impeding the return of the venous blood from the retina as by acting as an irritant on the tissues, the cerebro spinal fluid being loaded with toxic products of the tissue-change of the neoplasm.

In each of the foregoing theories the increased intra-cranial pressure is the primary factor in the causation of the papillitis. But there are those —Gowers,[5] Brailey,[6] Walter Edmunds,[7] S. Mackenzie,[8] etc.—who hold that this increased pressure has no such relation to the optic neuritis, which they believe to be nothing more than a descending neuritis transmitted from the intra-cranial cavity. These observers consider their view to be supported by the microscopical appearances of the optic nerve, and believe that the inflammation travels down the nerve from a meningitis, or cerebritis, set going by the irritation of the new growth. Those who maintain the increased pressure theory look upon an inflammatory process found in the stem of the optic nerve as an ascending, and not a descending, process, and regard both meningitis and cerebritis, except in the immediate neighborhood of the new growth, as rare in these cases.

The hypothesis which seems to Hughlings Jackson[9] to be the most plausible is the one put forward by Benedikt,—namely, that the papillitis is the result of reflex vaso-motor action. But it is doubtful if vaso-motor alterations are capable of producing inflammatory changes.

It must be admitted that a completely satisfactory theory of the causation of congestion papilla has not yet been propounded.

*Treatment.*—It need hardly be stated that little can be done by treatment in the vast majority of cases of optic neuritis due to cerebral tumor.

In all cases where the pathological diagnosis inclines to that of a syphilitic gumma, an active mercurial course is clearly indicated, and with reasonable hope of a satisfactory result in many instances.

Some years ago the cure of optic neuritis in cases of cerebral tumor by tapping of the sheath of the optic nerve to evacuate the dropsy was proposed by de Wecker and attempted by Brudenell Carter,[10] but the results

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[1] Archiv für Ophthalmologie, xv. 2, S. 193.
[2] Ibidem, vol. xvi pt. 1, p. 265.
[3] Transactions of the International Medical Congress, 1881.
[4] Ueber Neuritis Optica, Jena, 1887.
[5] Diseases of the Brain.
[6] Transactions of the International Medical Congress, 1881.
[7] Transactions of the Ophthalmological Society of the United Kingdom, i. p 112.
[8] Brain, ii. p. 257.
[9] Transactions of the Ophthalmological Society of the United Kingdom, i. p 90.
[10] Brain, x. p. 199.

do not seem to have been encouraging, and the method has not been adopted.

Trephining the skull is a measure to be recommended in many of these cases. Victor Horsley has pointed out[1] that even when the cerebral tumor cannot be removed, the effect of opening the skull is often to relieve the headache and arrest the optic neuritis; and in cases of tubercular tumor, which sometimes undergoes retrogression, with recovery of the patient, blindness may be averted. In other cases, where recovery cannot be hoped for, trephining may sometimes be indicated, with the object of retaining sight during the months of life which perhaps remain to the patient, and for the relief of the headache. In a case of cerebral tumor, with severe headache, intense optic neuritis, and almost complete blindness, exploration revealed a rapidly growing tumor on the temporo-sphenoidal lobe, quite beyond the possibility of removal. Nevertheless, as a result of the operation, the patient lived for several months free from pain and with recovery of sight, so that he was enabled to enjoy life to some degree. In the majority of cases, Horsley states, the neuritis subsides within three weeks after the operation. Bruns[2] and others have had similar good results after trephining in cases of cerebral tumor where the tumor was not removed.

Lumbar puncture, first proposed by Quincke[3] for the reduction of intra-cranial pressure in hydrocephalus, has been tried by Burchardt[4] in one case of congestion papilla with some degree of success.

2. **Attacks of Temporary Total Loss of Sight.**—These form a symptom of intra-cranial tumor which is often present, both with and without optic neuritis. The symptom used to be sometimes known as epilepsy of the retina, or as epileptiform amaurosis (Hughlings Jackson), but it is not thereby implied that it occurs in these cases in connection with an epileptic seizure, and the terms have now been abandoned. The patient may be able to read fine print, and there may be no derangement of the field of vision, and yet suddenly complete and absolute blindness of both eyes may come on, and may last from a minute or two to half an hour, the attack being liable to recur several times a day. It is probable that these attacks are due to some disturbance of the cerebral circulation affecting the functions of the visual centres or paths, or they may sometimes be due to a sudden increase of the cerebro-spinal fluid in the third ventricle, by which pressure on the optic commissure is brought about. It should be remembered that transient blindness occurs under several other conditions.

In cases of intra-cranial tumor the pupils are usually dilated, but they may be of normal size, and more rarely they are contracted.

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[1] Transactions of the International Medical Congress, Berlin, 1890, and British Medical Journal, December 23, 1893.

[2] Neurologisches Centralblatt, 1894, S. 20.

[3] Ibidem, 1891, p. 284.

[4] Centralblatt für Augenheilkunde, 1896, S. 313.

## LOCALIZING EYE-SYMPTOMS OF FOCAL DISEASE OF THE BRAIN.

The discussion of localizing symptoms in this chapter must be confined, for the most part, to the effects of lesions which we shall suppose to be limited to the part or region of the brain under consideration; yet in practice it is rarely that lesions are so limited. We commonly find them extending over more than one centre, and involving perhaps several paths, so that the symptom-picture resulting is a complex one, made up of the symptoms caused by the derangement of the functions of, it may be, two, three, or more cerebral regions. In such cases it becomes the duty of the physician to form for himself out of the symptoms presented, and by a consideration of the order of their onset, of the intensity of each of them, and so on, an opinion as to the position of the lesion which gives rise to them, and he must bear in mind the danger, so often unavoidable, of taking a distant symptom for a direct symptom. So far as is possible, however, the writer will endeavor to point out the concomitant symptoms which are most apt to be present with lesion of the part under immediate consideration.

For the sake of clearness, it is desirable to consider these localizing ocular symptoms under two heads,—A. Derangements of Vision, and B. Derangements of the Oculo-Motor Apparatus.

**A. Localizing Derangements of Vision caused by Focal Brain-Disease.**—It is proposed to discuss these symptoms according to the position of the presumed lesions, in the following order: (*a*) visual symptoms caused by lesions in the occipital lobe, cortical and subcortical; (*b*) visual symptoms caused by lesions in the posterior third of the posterior limb of the internal capsule; (*c*) visual symptoms caused by lesions of the primary optic ganglia; (*d*) visual symptoms caused by lesions of the optic tract; (*e*) visual symptoms caused by lesions of the optic commissure; (*f*) visual symptoms caused by lesions of the intra-cranial portion of the optic nerve.

*a.* Derangements of Vision caused by Lesions, Cortical and Subcortical, in the Occipital Lobe.—These symptoms are: hemianopsia, mind-blindness, amnesic color-blindness, visual hallucinations, and, when the lesion is on the left side of the brain in a right-handed person, word-blindness, visual aphasia, and dyslexia. These are the only visual symptoms, so far as we know, to which lesions of the occipital lobe give rise. This lobe, in short, seems to be given up entirely to the functions of vision in one form or another. But each of these functions is not spread out over the whole lobe; different parts of it would appear to be functionally specialized for special uses in the processes of seeing and recognition. Most important of all is the actual visual centre; and we shall proceed to consider the symptoms indicating a lesion of the cortical centre for vision.

There is now a mass of evidence which points to the cortical centre for vision being situated on the mesial surface of the occipital lobe, in the

cuneus or its immediate neighborhood, rather than in the angular gyrus or occipito-angular region, as held by Ferrier.[1] Among the earliest authorities to put forward the former view were Seguin,[2] Hun,[3] who indicated the cuneus in particular as being the centre for sight, Haab,[4] Féré,[5] etc., while the most recent and most important researches in this direction are those of Henschen,[6] of Upsala, of Vialet,[7] of Paris, and of Flechsig,[8] of Leipzig, set forth in their marvellously interesting monographs. Henschen, as the result of his clinico-pathological investigations, confines the cortical visual centre to the region immediately adjoining the middle part of the calcarine fissure. Moreover, he regards the upper or cuneic lip of the fissure as representing the homonymous dorsal retinal quadrants of the retinæ, while the lower or lingual lip he believes to represent the homonymous ventral quadrants of the retinæ. He believes that the macular centre is in the floor of the calcarine fissure. Vialet does not admit that the visual centre is confined to such narrow limits. He takes for it the whole mesial surface bounded anteriorly by the parieto-occipital fissure, above by the supero-mesial margin of the hemisphere, below by the inferior margin of the third occipital convolution, and behind by the occipital pole. This region corresponds with the distribution of the band of Vicq-d'Azyr, which Vialet inclines to think has to do with the sense of sight. He admits, however, that in this region the calcarine fissure has a very special importance. It is extensive and deep, nowhere else is the band of Vicq-d'Azyr so well developed, the fissure has a rich arterial supply, and the early appearance of its anterior and of a portion of its middle part in the fœtal brain indicates its importance. Yet Vialet's three beautifully recorded cases do not really refute Henschen's view; indeed, they seem rather to give it support, for in each of them the middle part of the calcarine fissure was diseased.

Flechsig believes with Henschen that the calcarine fissure corresponds specially with the macula lutea; but he holds, moreover, that the visual centre extends to the entire cuneus and the lingual lobe. His opinion is based on the investigations into which he has been led by his discovery of the gradual myelization of the nerve-fibres in the white substance of the brain in the fœtus and new-born infant, a discovery the importance of which it is impossible to exaggerate. Until the fifth month of intra-uterine

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[1] Diseases of the Brain, second edition.

[2] The Journal of Nervous and Mental Disease, 1886, p. 1, and Archives de neurologie, 1886, p. 176.

[3] The American Journal of the Medical Sciences, January, 1887.

[4] Klinische Monatsblätter für Augenheilkunde, 1882, S. 149.

[5] Archives de neurologie, 1885, p. 229.

[6] Klinische und anatomische Beiträge zur Pathologie des Gehirns, i. und ii., 1890, iii., 1892, i., 1894.

[7] Les centres cérébraux de la vision, Paris, 1893.

[8] Neurologisches Centralblatt, 1895, S. 1118, und 1896, S. 2. Localisation der Geistigen Vorgänge, Leipzig, 1896; Gehirn und Seele, Leipzig, 1896.

life the cerebral nerve-fibres consist only of axis-cylinders. At that period they commence in a regular order to be clothed with medullary sheaths, so that, knowing the age of a fœtus, it is possible to tell in what region the nerve-fibres would be found already provided with medullary sheaths, and in what region they would still be mere axis-cylinders. Flechsig also ascertained that certain bundles of fibres which have certain anatomical connections, and presumably, therefore, certain physiological functions, obtain their myeline sheaths at one period; while other bundles with different anatomical connections, and hence different physiological functions, obtain their sheaths at another period. By this method he has been able to distinguish between many of the most important paths of the brain, and to trace them to their centres.

But, while the question whether the cortical centre for vision is confined to a part of the calcarine fissure or occupies a more extensive region of the mesial surface of the brain is one of eminent interest, it is, for practical purposes, sufficient to know that this centre is round about the cuneus and the calcarine fissure, that it does not occupy the whole of the occipital lobe, and that it is not on the outer surface of the brain.[1]

The most important symptom caused by a destructive lesion of the whole of the cortical visual centre in one hemisphere is complete and absolute loss of function in the corresponding side of each retina, and, consequently, complete and absolute homonymous hemianopsia of the opposite side of each field of vision. But homonymous hemianopsia may also be caused by a lesion anywhere in the visual path between the centre and the optic chiasma, and it is important to be able, if possible, to decide, in a given case of hemianopsia, whether the lesion is cortical or in the visual path, and, if in the latter, in what part of it.

In cases of hemianopsia due to a lesion of the cortex alone we are mainly guided in our localization by negative signs,—the complete absence, namely, of any concomitant paralytic symptom, such as hemiplegia, hemianæsthesia, or aphasia, and also of mind-blindness, word-blindness, visual aphasia, hallucinations, and the hemiopic pupil. The one positive sign of this cortical lesion is, oddly enough, negative vision,[2] the "vision nulle" of Dufour.[3] The patient in these cases sees "nothing" in

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[1] Déjerine and Vialet recorded an important case (Société de biologie, Paris, December, 1893). It was that of a man who suddenly became blind of both eyes, without apoplectic seizure or loss of consciousness. The fundi oculorum and refracting media were normal, and the diagnosis of cortical blindness from bilateral lesion of the mesial surfaces of the occipital lobes was made. The pupil reflexes to light and on convergence remained healthy. The patient lived for a long time afterwards, and ultimately died of pneumonia. The post-mortem examination confirmed the diagnosis of the cause of blindness. On the right side the lesion occupied the cuneus and the lingual and inferior occipito-temporal lobes, and on the left side the lingual and inferior occipito-temporal lobes. Here again, as well as can be learned, the calcarine fissure is involved in the lesion on each side.

[2] Michel, Lehrbuch der Augenheilkunde.

[3] Revue médicale de la Suisse Romande, August 20, 1889.

the defective part of the field; he is, in fact, as unconscious of his defective sight in half of his fields as a healthy person is of the existence of the normal blind spot. On the other hand, if the lesion be in the visual path, a subjective sensation of darkness or of blackness is referred to the defective halves of the field of vision, and in these instances the patients frequently call the attention of the surgeon to their defect.

But a case published by Bleuler[1] renders it doubtful whether this symptom of negative vision can invariably be depended upon as indicating a lesion of the visual cortex; for, the author states, the symptom was present in that case, although the cortex was uninjured, the hemianopsia being caused by disease of the anterior part of the visual path, and a similar case has been seen by Wilfred Harris.[2]

It is rarely that lesions are confined to the cortical centre for vision. They are apt, at the least, to involve the medullary substance immediately under the cortex. They often, also, occupy a greater extent of the cortex on the mesial surface of the brain than that assigned to vision, or they involve the tip and outer surface of the occipital lobe, or, striking deeply into the medullary substance, they implicate the optic radiations and the commissural fibres to other centres.

It is strongly held by Henschen (see above) and others, but denied by von Monakow[3] and Vialet, that a correlation exists between parts of the retina and parts of the visual centre, and that it is possible for a lesion to be so situated in the visual centre as to cause blindness of only half of the homonymous half-field—in other words, of homonymous quarter-fields—of each eye. The most important case in proof of this is that published by Hun and already referred to. Here a lesion limited to the lower half of the right cuneus and the upper lip of the calcarine fissure caused a loss only of the left lower quadrant and of the peripheral portion of the left upper quadrant in each field. Hun concluded from this case that the fibres from the right upper quadrant of each retina terminate in the lower half of the right cuneus. The same, of course, would hold good for the left upper retinal quadrants and the right lower half of the left cuneus. A study of recorded cases seemed to Hun to show that the fibres from the lower retinal quadrants terminate either in the upper half of the respective cunei or in the lingual gyri, and he inclined to the latter position. In the light of Henschen's more recent and more extensive investigations, much credit is due to Hun for the sagacity of his conclusions.

A good many cases of double cortical hemianopsia are recorded.[4] By

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[1] Archiv fur Psychiatrie und Nervenkrankheiten, xxv. S. 32.

[2] Brain, Part lxxix., 1897.

[3] Archiv für Psychiatrie und Nervenkrankheiten, xxiv. S. 251.

[4] Bouveret (with post mortem), Revue générale d'Ophtalmologie, November, 1887; Förster, Archiv für Augenheilkunde, xxxvi. 94; Schweigger, Archiv für Ophthalmologie, xxii.; Groenouw, Archiv für Psychiatrie und Nervenkrankheiten, xxiii. 339; Vorster, Allgemeine Zeitschrift für Psychiatrie und psychisch-gerichtliche Medicin, xlix. 227; Schmidt-Rimpler (with post-mortem), Archiv für Ophthalmologie, xxii. 313; Déjerine

this term is meant loss of the homonymous half-field of each side, left and right, due to a lesion in the cortical centre for vision in each hemisphere, the complete result being, of course, blindness in the whole of the field of each eye. Most commonly one visual centre has become diseased some time before its fellow, the usual homonymous hemianopsia being produced, and subsequently a lesion of the opposite visual centre has destroyed vision in the remaining homonymous half-fields. In several of the recorded cases there was no post-mortem, and the diagnosis was made during life merely by the absence of all symptoms except the hemianopsia. In Schmidt-Rimpler's case both the symptoms and the autopsy showed the presence of diffused and disseminated cerebral lesions, rendering it impossible to assign the immediate cause of the visual symptoms. In Bouveret's and in the Déjerine-Vialet case already mentioned (foot-note, p. 548) the post-mortem examination displayed lesions occupying the mesial surface of each occipital lobe. It is interesting to note that in Förster's, Schweigger's, and Magnus's cases a small central portion of the field corresponding to the macula lutea was left intact.

In several of these cases (Förster, Groenouw, Schmidt-Rimpler, Vorster, Magnus, Dunn, Kaestermann), after the second hemianopsia occurred, the power of orientation, or the topographical sense, became more or less defective, so that the patient could not find his way about a well-known room or house, nor describe the relative positions of windows and doors that he had known for years, yet there were no other symptoms of mind-blindness. In Dunn's case, the patient had no conception of where he was, nor could he form a mental picture of the geography of his own house, or of any place he had ever been to. He could recollect that he lived at the corner of two streets, and their respective names, but their relation to each other, or to other streets, was completely lost. When a place was named he could remember it, but he could not call up its locality, or its relation to other places. He still was able to describe correctly the appearance of individuals. Although he retained a small central field in each eye, and had for two years taken almost daily drives or walks about the town with an attendant, yet he regained nothing of his lost topographical sense, and was quite incapable of going a short distance, or even about his own room, alone. The absence of this symptom in such cases until the second hemianopsia occurred seemed to be reasonably accounted for, at first, by the assumption that, as regards the function of orientation, the cortex of the healthy hemisphere acted for that of the diseased one. But that this is not

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and Vialet (with post-mortem), Centralblatt für Augenheilkunde, February, 1894. S. 63; Swanzy and Werner, Transactions of the Ophthalmological Society of the United Kingdom, 1891; Magnus, Deutsche medicinische Wochenschrift, January 25, 1894; Allyn, Archives of Ophthalmology, xxv. p. 206; Peters, Archiv für Augenheilkunde, xxxii. S. 175; Dunn, University Medical Magazine, Philadelphia, May, 1895; Anton (with post-mortem), Mittheilungen der Verein der Aerzte in Stiermark, 1896, Nr. 3; Brückner, Inaugural Dissertation, Giessen, 1896; Kaestermann, Monatsschrift für Psychiatrie und Neurologie, Band ii., 1897 (with post-mortem).

the correct explanation became evident from Peters's case, in which, immediately on the occurrence of the first (left) lateral hemianopsia, marked loss of the topographical sense manifested itself, and continued until the fatal termination of the case, three years later, without the second (right) lateral hemianopsia, when it came on, aggravating the symptom in any way. Peters's case taken alone might point to the right hemisphere as possessing a greater influence over the power of orientation than the left, but that this cannot be so is proved by Groenouw's case, in which the loss of this power did not take place until the left hemisphere was attacked. Peters infers that the loss of the topographical sense is not due to a cortical or subcortical lesion, but rather to damage to association paths between certain centres by the combined action of which the function of orientation is effected; and both in his own and in Förster's case there were lesions extending deeply into the white substance. From Peters's case it is obvious that the symptom under consideration might occur in a case of simple lateral homonymous hemianopsia, although the writer is not acquainted with any such case as yet on record.

Lesions of the visual centre, as also, indeed, of the visual path, may not cause hemianopsia in the strict sense of the term, nor yet loss of half of the half-fields as above described, but rather partial irregular homonymous defects in the fields, most frequently at their peripheries, but sometimes in the form of scotomata at or near the centre of the fields.

As regards peripheral defects in the fields, it is necessary to observe that in nearly all recent focal brain-diseases small homonymous peripheral contractions of the fields are very common, especially with tumors and abscesses, and they are also sometimes found in very debilitated patients, without any lesion in the visual centre or path: hence caution must be exercised in utilizing such defects for localization. Again, in hemianopsia we often meet with concentric contraction of the seeing half-fields, especially with extensive lesions, even of only one hemisphere; and this, too, is very common in cases of recurring apoplexies and after epileptic attacks. The contraction is probably due to inertia of the visual centre or of the retina. (See also p. 569.)

In the foregoing we have been treating of complete hemianopsia,—that is, the form of hemianopsia in which the whole of one side of the field of vision, with the exception, perhaps, of the fixation point, is wanting,—and of incomplete or partial hemianopsia,—that is, the form in which a sector only of the half-field is wanting. In each instance the hemianopsia was assumed to be absolute, including all the three visual perceptions,—color, form, and light.

But cortical hemianopsia may be relative,—that is, the lesion may destroy only the color-sense in the half-field (hemiachromatopsia), leaving the form-sense and light-sense intact, or it may destroy the color- and form-senses, leaving the light-sense intact. Cases, moreover, are observed in which in one part of the defective field all three senses are lost, while in

the remainder only the color- and form-senses are wanting, the light-sense being retained. All three senses, too, may be reduced in degree over the entire half-field, without complete loss of any of them. Sometimes the color sense is lost, while the form- and light-senses are only greatly reduced in degree; and in such cases the color-blindness in the half field is probably due to the reduction in the light-sense. Recorded cases render the theory plausible—but nothing more—that in the cortex the centre for the color-sense is differentiated from the centres for the form- and light-senses, and also that the different colors, or at least the different primary colors, have special cells set apart for their use. Henschen, for example, records a case of cortical hemianopic violet blindness, the power of distinguishing all other colors remaining good in the same half-field. Defect of the color-perception in the half-fields with lesions of the optic tract or of the chiasma has been observed, and will be referred to again.

The explanation of the occurrence of relative cortical hemianopsia has excited much attention. Wilbrand's attractive theory,[1] that the color-sense, form-sense, and light-sense are arranged in layers over each other in the gray matter in the order named, from without inward, is as yet unsupported by sufficient evidence; and it would be difficult to conceive of a lesion so nicely situated as to destroy, say, the outermost layer of the cortex, but to leave its deeper parts untouched. The view is indeed now gaining ground that there are not separate cortical centres, layers, or cells for color, form, and light, but rather that relative cortical hemianopsia is a manifestation of a lesion of less intensity than that which causes absolute hemianopsia. In Verrey's[2] and in Mackay's[3] cases, as well as color-blindness, there was diminution, but no absolute loss, of the light- and form-senses in the half-field; and Mackay points out that in the other recorded cases of hemi-achromatopsia sufficient care was not taken to investigate the form-sense in the affected half-fields. Moreover, it must be admitted that non-cortical lesions—even those, as stated above, which are situated in the chiasma—may produce hemiachromatopsia. This view, then, would render the loss of function in hemiachromatopsia analogous to that which is found in the central scotoma of commencing toxic amblyopia. Vialet is among those who are opposed to the theory of Wilbrand.

Homonymous lateral hemianopsia, as Gowers and other authors have pointed out, very frequently occurs as a distant symptom in cerebral apoplexy, without regard to the position of the hemorrhage. In the early stages of the case, if the surgeon's hand be brought suddenly before the eye, first from one side and then from the other, it will be found that the eyelids blink when the hand comes from the unparalyzed side of the body, but not when it comes from the affected side. This hemianopsia usually

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[1] Ophthalmiatrische Beiträge zur Diagnostik der Gehirnkrankheiten, 1883.

[2] Archives d'Ophtalmologie, Juillet-Août, 1888.

[3] British Medical Journal, November 10, 1888.

passes away in a few days. The writer has seen it last three weeks. It is part of the transient symptoms of the apoplectic seizure, and has no localizing value whatever. As a distant symptom, hemianopsia is not common with other forms of cerebral lesion, although it is now and then so met with. In attacks of Jacksonian epilepsy, homonymous hemianopsia is not rare.

It is now generally conceded that the macula lutea is specially represented in the cortical centre; but there are two views as to the arrangement of this macular centre. These views have been called into existence by the desire to explain the fact that in hemianopsia the line of demarcation sometimes passes through the fixation-point in the field, but more commonly leaves it in the seeing half. In these latter cases the dividing line lies in the vertical meridian until within eight or ten degrees both above and below the fixation-point, where it commences to curve round that point, and keeps at about the same distance from it towards the blind side of the field, a bite being taken, as it were, out of the latter. Sometimes, again, the line of demarcation lies in its whole length some five to ten degrees removed from the vertical meridian towards the blind side of the field, and thus a whole stripe is added to the seeing half of the field.

According to one view of the macular supply, which is supported by Wilbrand, Henschen, and Gowers, the whole of the macular region of each retina is innervated from the macular centre of each hemisphere; there is, in short, an overlapping of nervous supply to these retinal regions. Consequently, if there be a lesion at the visual centre, including the macular centre, in one hemisphere, that in the other hemisphere being sound, the functions of the whole of each macula will be preserved. Cases where occasionally in cortical lesions the line of demarcation in the field does go through the fixation-point would be accounted for, under this theory, by an individual variation in the supply of the maculæ, which in these instances would be similar to that of the remainder of the retinæ.

According to the other theory, which is put forward by Wernicke,[1] Förster,[2] and Schweigger,[3] the macular region of the retina is invariably supplied on the same plan as the rest of the retina,—*i.e.*, each side of it from the corresponding side of the brain. In order to explain why it is that in some cortical lesions the line of demarcation passes through the fixation-point, while in others it deviates at this place towards the blind side, the supporters of this view state that the cortical centre for the macular region is more richly supplied with blood-vessels than the rest of the visual centre. Hence, when the lesion is an embolism or thrombosis of the vessels supplying that part of the brain, this special region, by reason of abundant anastomoses, preserves its functions. But if the lesion is a hemorrhage, the macular region of the cortex is apt to be involved in

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[1] Lehrbuch der Gehirnkrankheiten.

[2] Archiv für Ophthalmologie, xxxvi. S. 94.

[3] Archiv für Augenheilkunde, xxii.

the lesions with the rest of the visual centre, and loss of function in the corresponding half of the macula lutea, with the line of demarcation passing through the fixation-point, is the result.

It seems to the writer that each of these hypotheses is defective; for, to be satisfactory, any such theory must be capable of explaining the phenomenon in question, not only when the lesion is in the cortex, but also when it is in the tract or chiasma. Yet, according to the first theory, in lesions of the tract or of the chiasma the fixation-point would always be spared; and according to the second theory, these lesions would always cause the dividing line to pass through the fixation-point. As a matter of fact, however, with lesions at either of these situations, just as with lesions in the occipital lobe, the dividing line sometimes passes through the fixation-point and sometimes to one side of it.

Von Monakow[1] puts forward an elaborate theory to account for the frequent immunity of the fixation-point, in which the basal ganglia play an important part, and if Flechsig's[2] observation that the macula lutea is directly connected with the external geniculate body only, and not with the anterior quadrigeminal body or optic thalamus, be correct, that fact may be found to have an important bearing on the point under consideration. Schmidt-Rimpler[3] is of opinion that the varieties in the line of demarcation are due to individual varieties in the distribution of the retinal fibres in the tract. For the most part, one-half of the retina is supplied by one tract, the other half by the other tract, but without, he thinks, this supply being always arranged with mathematical precision in respect of the dividing line. Finally, there are some ophthalmologists who hold that the line of demarcation does in fact always pass through the fixation-point, and that it is merely imperfect fixation on the part of the patient during the perimetrical examination which makes it seem to pass round it. This is certainly incorrect.

The inconvenience caused to patients by the loss of one side of the field of vision is considerable, but, as the fixation-point is usually spared, fairly good, although, as a rule, somewhat impaired central vision, as also the power of reading, etc., is retained. Right hemianopsia gives rise to difficulty in reading, which does not belong to left hemianopsia. This is believed to be because we read from left to right, and in order to do so fluently it is necessary to be able to catch up rapidly the word or two immediately following that which is being looked at; and this process becomes difficult or impossible in this hemianopsia, owing to the loss of the right side of the field. Knies offers a different solution of this difficulty, at least for cases due to lesions in the occipital lobe, which will be mentioned later. (Vide p. 585.)

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[1] Loco citato.

[2] Gehirn und Seele, S. 71.

[3] Archiv für Augenheilkunde, xxvi S. 190.

Horizontal cortical hemianopsia, superior or inferior, might conceivably be caused by symmetrical lesions in each hemisphere; but as yet no such case is on record, at least none which could with any degree of certainty be regarded in that light.

*Visual Symptoms indicating Lesions of the Optic Radiations.*—Here, too, homonymous lateral hemianopsia is the great symptom. It is commonly supposed that all these lesions have homonymous lateral hemianopsia as a constant symptom, the optic radiations being regarded as the visual path. According to Henschen, this is but partially true, for his researches have led him to conclude[1] that the visual path occupies only the central portion of the optic radiations, as the latter term is anatomically understood, and that it there forms a bundle less than a centimetre thick, which runs in a curve round the posterior horn of the lateral ventricle, on its outer side, at the level of the second temporal convolution, to reach the calcarine fissure. Flechsig, too, states that, as the optic radiations contain at least five times as many fibres as do the optic nerves, it is obvious that there must be paths in the optic radiations which have nothing to do with the conduction of visual stimuli. Furthermore, a case of Anderson's[2] and one of Bruns's[3] show, in Henschen's opinion, that the fibres for the upper half of the half-retina run dorsally in the visual bundle, and those for the lower half of the half-retina ventrally in the visual bundle. Vialet adopts the whole of the optic radiations as the visual path. It seems desirable to give Vialet's statement of the course of the optic radiations in the occipital lobe according to his anatomical investigations and his study of secondary degenerations in these fibres. He says, "The fibres which emanate from the cuneus follow two different courses. Those which proceed from its upper region pass over the forceps major in turning round the upper wall of the posterior horn of the lateral ventricle. Those which proceed from the lower part of the cuneus unite with the fibres coming from the calcarine fissure and lingual lobe, and pass obliquely from above downward and from behind forward, taking a spiral course round the inferior wall of the posterior horn, and join together on the external wall of the latter. The fibres which emanate from the inferior occipito-temporal convolution divide into two portions: those of the smaller section, coming from the inner half of the convolution, unite with the fibres from the calcarine fissure and lingual lobe, and follow the same path; those of the larger section, coming from the outer half of the convolution, proceed either along the under wall or along the external wall of the ventricle. Finally, the fibres which come from the apex of the occipital lobe proceed directly along the external wall of the posterior horn."[4]

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[1] Brain, Spring Number, 1893; Beiträge zur Pathologie des Gehirns, ii.

[2] Transactions of the Ophthalmological Society of the United Kingdom, x. p. 243.

[3] Neurologisches Centralblatt, 1890, S. 508.

[4] On this subject see also Brissaud, Annales d'Oculistique, November, 1893, and Vialet, ibidem, March, 1894.

It is, the writer thinks, important to mention here that a few cases of abscess of the brain resulting from purulent otitis are published in which homonymous hemianopsia was present. In one recorded by Lannois and Jaboulay[1] the abscess was in the occipital lobe. In a case successfully operated on by R. H. Woods[2] at the Richmond Hospital, Dublin, the condition of the patient prior to the operation did not admit of an examination of the functions of vision. After recovery the only symptom left was a defect of vision, which the writer ascertained to consist in incomplete right homonymous hemianopsia, due, no doubt, to implication of the visual path in the left temporo-sphenoidal lobe, where the abscess had been situated. These cases show how important it is, before operation, to aid the local diagnosis of cerebral otitis abscesses by an examination of the field of vision whenever possible.

The clinical diagnosis between a lesion in the cortical centre for vision and one in the optic radiations, or in the visual bundle of these radiations, where the lesion implicates only one or other of these parts, may perhaps be made. With lesions in the optic radiations hallucinations might be present, but not with cortical lesions. With the former there would be the subjective sensation of blindness in the defective side of the field, while with the latter there would be "vision nulle" in the defective half-fields. When the lesion in the medullary substance involves a wider extent, other paths than that for vision become implicated, and then collateral symptoms are present, such as mind-blindness, with the lesion in either hemisphere or both hemispheres, or word-blindness, visual aphasia, etc., with the lesion in the left hemisphere, and if the disease extend forward towards the internal capsule there may be hemiparesis or hemiplegia. These symptoms will now be considered in greater detail.

*Mind-Blindness, or Visual Amnesia.*—According to the prevailing theory of the process of visual recognition, there is in the cortex of the occipital lobe, besides the centre for vision, a centre for visual memory. In the cells of this centre there are believed to be stored up the impressions or visual memory-pictures of scenes and objects, images of which have from time to time been formed on the retina and perceived by the centre for vision. When an object is looked at which, or the like of which, the individual has seen before, the impression conveyed to the centre for vision is carried on to the visual memory-centre, is there compared with the corresponding visual memory-picture, and recognition takes place. Or if the object be one which the individual has never before seen, its memory-picture is stored up for future use in his visual memory-centre. The faculty of visual memory varies a good deal in different persons, according as they are specially endowed or as education and use develop the power, and it is

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[1] Gazette médicale de Paris, 1896, No. 37.

[2] Transactions of the Royal Academy of Medicine in Ireland, November 12, 1897; British Medical Journal, January 22, 1898.

present more for certain classes of objects than for others in some persons, and more than in other people. The vividness, and even the existence, of a visual memory-picture depend largely on the degree of desire to retain such an impression of it which accompanies the reception of its image on the retina and in the centre for vision. It is possible to look at an object and yet not retain even an ephemeral impression of it in the visual memory-centre, no desire to do so or ground for doing so being present. On the other hand, we look at some objects or sights with more or less interest and with an effort of the will to obtain and retain visual memory-pictures of them, and these pictures can often be utilized in recognition, and can also be called up in our fancy—in our "mind's eye"—after long intervals. Sights, too, which produce strong emotions (joy, pleasure, sorrow, horror) are frequently impressed deeply on our visual memory.

The theory of a centre for visual memory more or less distinct from the centre for vision was first propounded by Munk, who, in his experiments on dogs,[1] observed the symptom which he named "Seelenblindheit" (mind-blindness). On extirpation of a certain region of the occipital lobe of a dog, he found that, although all the senses, including sight as well as motion, were unimpaired by the operation, the animal no longer knew his master, of whom he had been fond, nor his dog friends. When hungry and thirsty, he did not look for his food where he had always found it, nor did he find the food placed close to him, unless he smelt it. He saw the whip with which he had sometimes been punished held menacingly at him without evincing fear. He had been taught to give the paw when a hand was held towards him, but this he now no longer did, until the word "paw" was called to him, and so on. That he was not blind in the ordinary sense of the word was shown by the fact that he ran about freely, jumping over, creeping under, or running around obstacles when they came in his way. The dog had only lost all his memory-pictures; he was, in respect of visual memory, where a new-born puppy is.

It was soon observed that a similar symptom is sometimes seen in man with some focal lesions of the occipital lobe and its neighborhood.

The symptom of mind-blindness occurring in man consists in loss of the power to recognize objects when seen only, although the acuteness of vision and the general intelligence may remain perfect, and the patient can read: there is no word-blindness. But if some one or more of the other senses be brought to bear on the object, the patient at once finds out what it is. A patient of the writer's, whose sight was good, could not recognize his friends when he saw them; he would pass his wife on the street, and when she would stop him, he would look her straight in the face, and would not recognize her until she spoke, but as soon as he heard her voice he would know who it was. Shown a needle, a person afflicted with mind-blindness could not say what it was until he took it in his hand and pricked

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[1] Ueber den Functionen der Grosshirnrinde, Berlin, 1881.

his finger, nor a watch until he heard it tick, nor a rose until he smelt its perfume.

Two kinds of mind blindness are recognized,—the cortical and the transcortical. In the former the lesion is in the centre for visual memory, while in the latter only the path connecting the centre for vision with the visual memory-centre is interrupted.

In cortical mind-blindness the memory-pictures themselves are lost. Here the patient is unable either to tell what an object is which he only sees, or to describe an object he is not looking at: visual memory-pictures can no longer be called up in his imagination. He cannot describe the appearance of his nearest relations, the arrangement of the furniture in his rooms, the well-known way from one part of the town to another, and so on; while, too, his relations and friends, when he actually sees them, are like strangers, he does not know his own face in the glass, he does not feel at home in his own house, he cannot find his way through his own town, nor tell the use of the most common objects which he only looks at.

In transcortical mind-blindness the patient is perfectly able to describe a well-known face, object, or place when he is not looking at them, but immediately afterwards, if shown any of these, he fails to know that they are what he has been describing. A patient of Wilbrand's,[1] for example, could, in her mind's eye, picture to herself the streets of the town in which she lived, the faces of her friends, and the arrangement of the furniture in her room, yet all these seemed strange to her when she looked at them; in fact, she could find her way about her room with greater comfort when she shut her eyes than when she kept them open. This form of mind-blindness is the more common of the two; but pure instances of either form are rare, most cases partaking mainly of the features of one form and to some extent of those of the other.

Hemianopsia, or at least a homonymous defect in the field of vision, is present in the majority of cases of mind-blindness. But it must not be taken for granted that in a case of mind-blindness with hemianopsia the lesion causing the former symptom is a one-sided one, for a lesion may be present in the centre for visual memory in the opposite hemisphere, or in its association paths, without involving the centre for vision or the optic radiations.

Color-blindness, in the form of amnesic color-blindness, or of true color-blindness, is not rare with mind-blindness, and the true color-blindness may either occupy the whole of each field, or be present as hemiachromatopsia.

The position of the visual memory-centre, and its anatomical relations to the centre for vision, are still to a great extent subjects for debate. Henschen[2] is of opinion that the lateral surface of the occipital lobe is the

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[1] Die Seelenblindheit, Wiesbaden, 1887.

[2] Loco citato.

seat for this function, while Wilbrand,[1] Nothnagel,[2] and others assign to it the whole of that lobe, except where the centre for vision resides, and Gowers[3] places it in or about the angular gyrus. According to Flechsig,[4] mind-blindness (with other symptoms, such as mind-deafness, mind-anæsthesia, weakening of visual ideation, incapacity to call to mind formerly well-known melodies, etc., and, in certain extreme cases, idiocy with incoherence) is caused by lesions of his posterior great association-centre, which is situated between the visual, tactile (Rolandic region), and auditory (part of the exposed portion of the first temporal convolution, and that portion of the same convolution which assists in forming the operculum) centres, and corresponds with the area occupied by the precuneus, part of the lingual convolution, the fusiform convolution, all the parietal convolutions, the inferior temporal convolution, and the anterior part of the external surface of the occipital lobe. There is no doubt that the lesion has been found in the occipital lobe, and sometimes extending over to the parietal convolutions, in most cases of mind-blindness. In two-thirds of the cases both hemispheres were diseased, and it is not improbable that in the remaining cases a careful microscopical examination would have shown disease in each hemisphere. In short, it is for the present believed that a double-sided lesion is required to produce prolonged or permanent mind-blindness, but the possibility of a monolateral lesion causing it has not yet been definitely excluded. Recorded cases render it probable that monolateral lesions may at least give rise to a certain degree of mind-blindness, which passes away after a time.

It is right to mention that H. Sachs, in his interesting and important work,[5] does not favor the theory of a special visual memory-centre. Schmidt-Rimpler, too,[6] maintains a similar opinion.

All hitherto published cases of mind-blindness due to focal lesions have been in persons of middle age or older. In most of the cases the lesions consisted in softenings or hemorrhages, and the onset was consequently sudden; but occasionally tumors of the brain have been accompanied by this symptom. In focal lesions, then, mind-blindness is seen as a direct symptom, but yet it is probably more common as a distant symptom.

Besides its occurrence with focal lesions, mind-blindness is found in cases of general paralysis, usually in the advanced stage. It is also seen in some patients whose general state, and with it their mental energy, are much reduced by exhausting illness.

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[1] Loco citato.

[2] Die Localisation der Gehirnkrankheiten, Sechte Congress für innere Medicin zu Wiesbaden.

[3] Diseases of the Nervous System, i. p. 161.

[4] Loco citato.

[5] Vorträge über Bau und Thätigkeit des Grosshirns und die Lehre von der Aphasie und Seelenblindheit, Breslau, 1894.

[6] Die Erkrankungen des Auges in Zusammenhang mit anderen Krankheiten, S. 110.

Siemerling has shown[1] that mind-blindness may, to some extent, be artificially produced in healthy persons by causing them to look through greased spectacle-glasses, which reduce the acuteness of vision to about $\frac{1}{30}$, and by placing them in monochromatic light, so that they are deprived also of their color-vision. They are then unable to recognize many objects, although they can see them fairly well. Consequently, care must be observed in the diagnosis of mind-blindness, which should be made only if the vision is good enough to see objects plainly, and to enable the patient to go about with ease. At the same time, mind-blindness is usually accompanied by some diminution in the acuteness of vision, or by hemianopsia, or color-blindness. But it may be unattended by any loss of sight.

Mind-blindness is easily overlooked, for well-marked cases may be mistaken for true partial blindness, and the patients are apt to state to the consultant that they "cannot see" a face or an object, when their difficulty is not one of sight, but of power of recognition, while slight cases may escape observation in the same way that all slightly marked symptoms may do. Hence mind-blindness is probably more common than the number of cases of it on record would lead one to think.

*Word-Blindness, or Alexia.*—This symptom consists in loss of the power to read written or printed words, while the powers of understanding speech and of talking are retained. The patient can see the words perfectly, yet does not know what they mean nor how to pronounce them. To him they convey no more idea than if the page were printed in a foreign language that he had never learned. Although words cannot be read, yet, in most cases, letters may be read with more or less difficulty, but they cannot be put together into words, or, at best, some short, familiar words are picked out. Complete inability to recognize letters (literal alexia) is rare. Figures (numerals) can usually be read well. The patient cannot copy printed or written words, but he can write spontaneously or to dictation, although immediately afterwards he cannot read what he has written, not even his own signature. He can make out the writing, if he be permitted to go over the characters with the point of his pen. Other visual objects are named with ease (no visual aphasia), and their use recognized (no mind-blindness). There need be no motor nor amnesic aphasia, nor is the intelligence enfeebled. This is the "pure word-blindness" of Déjerine,[2] or the "subcortical alexia" of Wernicke.[3] There is also "word-blindness with agraphia" (Déjerine), or "cortical alexia" (Wernicke), in which, in addition to pure word-blindness, as above, the patient is unable to write spontaneously or to dictation.

Pure word-blindness is held to be due to an interruption in the path between the visual memory-centre and the speech-centre,—the centre for

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[1] Archiv für Psychiatrie, xxi. S. 284.

[2] Mémoires de la Société de biologie, Février, 1892.

[3] Lehrbuch der Gehirnkrankheiten, i. SS. 207, 388.

speech-sound memory; hence the sight of the word fails to awaken the memory of its speech-sound, and it can neither be spoken nor recognized. For as we associate the look of letters and words with their spoken sounds in learning to read, so it is necessary for the recognition of these symbols in after-life that each of these respective mental images—visual image and auditory image—should be capable of being awakened by a stimulus applied to either of them.

Flechsig[1] believes that injury of that part of his great posterior association-centre on the left side, which occupies the anterior portion of the second and third occipital convolutions, is capable of causing alexia.

The symptom of pure word-blindness has a localizing diagnostic value. In five cases with post-mortems which Bruns collected from the literature, the lesion was found in the left occipital lobe, and Déjerine[2] has gone further, and has defined the inferior part of the inferior longitudinal fasciculus in the left hemisphere, as the particular tract a lesion of which gives rise to pure word-blindness: hence this tract must be regarded as forming the path which connects the centre for visual memory (or the posterior great association-centre of Flechsig) with the speech-centre.

That literal alexia should not always attend on verbal alexia is an interesting circumstance, which requires an attempt at explanation. The most obvious one is, that the lesion of the path between the visual word-memory centre and the auditory word-memory centre, or speech-centre, is only a partial one, admitting of the simple process of naming and recognizing letters, but not of the more complicated process involved in the recognition of words; for the reading of entire words demands rapidity and continuity in the recognition of the letters composing them, as also the power of obtaining and retaining clear memory-pictures of those letters, so that no letter of a word may be forgotten before the end of the word is reached. Hence, for the reading of words, the integrity of the direct path between the two centres in question is, probably, of eminent importance, although for the naming of letters its perfect healthiness may not be necessary. Bruns[3] offers another explanation. He thinks letters may be made out by means of the muscle-sense through the eye-muscles, and by the connection between this sense, or its cerebral seat, and the speech-centre, the path then being visual memory, muscular sense, speech-centre. In this way the two attributes of letters—their form and their spoken sound—are obtained, and they are recognized and named.

It is conceivable that words, too, might with difficulty come to be read through this roundabout path; but, in view of their greater complexity, that is improbable.

The fact that numerals can, as a rule, be read in cases of alexia is not

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[1] Loco citato.

[2] Loco citato, and Vialet, loco citato, p. 295.

[3] Neurologisches Centralblatt, 1894, S. 57.

capable of a satisfactory explanation, other than that provided by the partial lesion theory. It may in some way depend on the fact that they are symbols which have a complete mental value independently of their speech-sound.

The other form of word-blindness, that with agraphia, is held both by Déjerine and Wernicke to be caused by a lesion in the centre for visual memory for words, which in right-handed persons is in the left angular gyrus. In a collection of cases made by Allen Starr,[1] he found that, when limited in extent and strictly cortical, the lesions producing this symptom were in the left angular gyrus, and in the cortex immediately anterior to it in the inferior parietal lobule.

Right homonymous hemianopsia is a very common concomitant symptom of word-blindness, as will be understood from the proximity of the visual centre and paths to some of the localities lesions of which cause word-blindness.[2] The difficulty in reading in some cases of right hemianopsia is doubtless due to the word-blindness present at the same time.

Word-deafness, more or less aphasia, and paraphasia will be found with word-blindness, should the lesion extend forward towards the temporal cortex. Word-blindness may be a distant symptom. Bruns[3] observed, in a case of recurrent apoplexy, the symptom to come on after each seizure and to last several weeks.

Word-blindness can hardly be regarded as partial mind-blindness, for in many marked cases of the latter it has been absent.

*Visual Aphasia.*—Freund has described[4] a symptom which he terms visual aphasia, and instances of it have since been recorded by others. It consists in the patient failing to find the names of objects seen, the use of which he knows, yet he can name those objects if, with closed eyes, he feels them with his hands. Moreover, this inability to name objects extends also to current speech, so that nouns are avoided, and paraphrases and perplexed expressions are substituted for them. In nearly all the cases observed there has been right homonymous hemianopsia, and many of them have been complicated with other symptoms, such as alexia and agraphia. Freund holds that this symptom is due to lesions in the left occipital lobe, occurring in the paths from the centres for visual memory in each hemisphere to the speech-centre in the left hemisphere. Varieties and degrees of the symptom probably depend on the position of the lesion as regards these several centres, and the accompanying diagram (Fig. 1) will assist in making this clear.

If, for example, there be a lesion at $x$, there will be right homonymous hemianopsia, and no new visual memories can be stored in $O^1$. Visual

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[1] Brain, July, 1889.

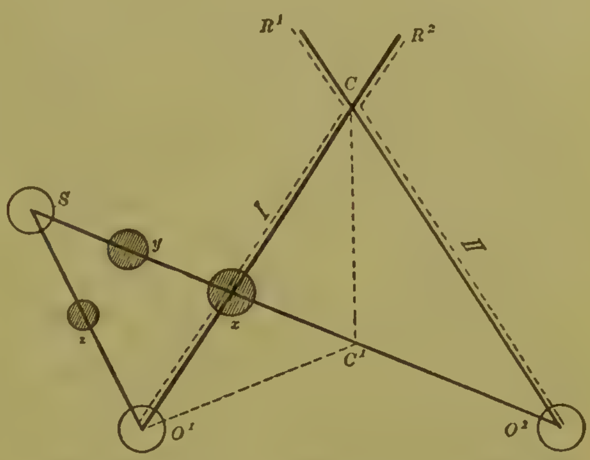
[2] See Byrom Bramwell, The Lancet, 1897, March 20, March 27, April 3, and April 17.

[3] Loco citato, p. 63.

[4] Archiv für Psychiatrie und Nervenkrankheiten, xx. S. 276.

memories which previously existed in $O^1$ are still present there and are not cut off from $S$, consequently they can be employed in speech. In $O^2$ old visual memories are intact and new ones may be acquired by it, but none of the visual memories which happen to be stored in this right centre can be employed in speech. In speech the patient will have a difficulty in finding nouns, in so far as they are expressions for new visual memories, or for such old ones as are stored in the right visual centre alone. He will know the uses of objects he looks at, but will be unable to tell their names. He will be able to point out correctly objects which are named to him, because, owing to the integrity of the paths from $O^2$ to the other sensory centres, he possesses an intact, concrete idea of the objects seen. Similarly, the patient is able, by means of his other senses, to name objects correctly. Again, if there be destruction of $O^1$, and in the path of $O^2$ $S$, say at $y$, the result will be: right hemianopsia, partial mind-blindness from loss of all visual memories stored in $O^1$; new visual memories can be stored in $O^2$, but are cut off from $S$. The use of objects, of which the visual memories have been stored in $O^1$ alone, will not any longer be understood, nor can they be named. Objects which have their visual memories in $O^2$ will be recognized as to their use, but cannot be named; but if named to him the patient will be able to point out objects correctly, and he can name objects by the aid of his other sens s. If there be lesions at $y$ and at $z$, there will be complete visual aphasia, for the patient will not be able to name any object shown him, nor in current speech to use any of the visual memories of objects stored in either visual centre. There will be no hemianopsia nor mind-blindness. By aid of the diagram, other modifications of the symptom which may occur in practice can be constructed, although, as yet, only some of them have been observed. The reader will note that Freund adopts a theory, not universally held, that the visual memory-pictures of objects are stored some in one hemisphere and some in the other; also, that he assumes a direct path from the right visual memory-centre to the speech-centre in the left hemisphere,—a path which, if the doctrine be true that commissural fibres between the hemispheres connect only identical regions, does not exist. But, although this direct path may not exist, yet the general principle of Freund's theory may be correct.

FIG. 1.




$S$, centre for speech; $O^1$, left visual and visual memory-centres; $O^2$, right visual and visual memory-centres; $R^1$, left retina; $R^2$, right retina; $C$ $C^1$, corpus callosum; $I$ and $II$, paths from the visual centres and visual memory-centres to the retinæ; $S$ is connected by a path with $O^1$ and $O^2$. Through the splenium at $C^1$ run the paths connecting the right visual centre and visual memory-centre with the speech-centre, and the visual centres and visual memory-centres with one another.

*Dyslexia.*—This symptom was first described by Berlin.[1] Like word-blindness, it belongs in a wide sense to the aphasia group. It consists in a want of power of the patient to read more than a very few words consecutively, either aloud or to himself. The difficulty is not caused by dimness of sight, nor by pain in the eye or head, but simply by an unconquerable feeling of dislike or disgust to this particular mental effort. After a few words, which can be well understood, have been read, the book is pushed away and the head is drawn backward and turned aside, and then in a moment or two the patient may be tempted to repeat the effort, but with the same result after very few words have been read. The symptom comes on suddenly, and has been usually the first sign of the presence of grave cerebral disease. Although, in most cases, the dyslexia disappeared in a few weeks, either permanently or to recur later, yet other symptoms soon followed its first onset, such as headache, giddiness, aphasia, hemianopsia, paralysis of the tongue, hemianæsthesia, hemiplegia, twitching of the facial muscles, etc., and the cases ended fatally. Atheroma of the cerebral vessels, followed by softening or hemorrhage, is the usual pathological condition present. The seat of the lesion is often, but not necessarily so, at or near the speech-centre. It may be in other parts of the left hemisphere, or in the right hemisphere, even in left-handed persons, and is of little value for localization. It is probably, indeed, for the most part a functional or distant symptom. Pick[2] points out that dyslexia is analogous, both clinically and pathogenetically, to painful intermittent lameness in horses,—a symptom which Charcot described as occurring in man also, and as having great clinical and prognostic importance. In the horse it indicates stenosis of the aorta, and in man—as Charcot showed—commencing obliteration of cerebral arteries.

*Amnesic color-blindness* is a symptom which has been described by Wilbrand.[3] It has nothing in common with derangement of the color-sense as such. In the cases observed it has always been accompanied by right homonymous hemianopsia, and is an indication, Wilbrand thinks, of focal disease in the occipital lobe interrupting the paths between the centre for color-vision, which he believes to exist, and the speech-centre. In amnesic color-blindness, although the patient can perform Holmgren's tests perfectly, yet he is unable to give to each color its name. He is either unable to give any name to the colors shown him, or he has only one term for all colors, or he describes colors by paraphrases or newly-formed words. He may also wrongly describe the color of familiar objects. On a superficial examination this symptom might be taken for true color-blindness, but it belongs, in fact, to the group of aphasia symptoms. The symptom of the same group with which it is most frequently associated is alexia.

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[1] Archiv für Psychiatrie und Nervenkrankheiten, xv. S. 275, and in his monograph, Eine besondere Art der Wortblindheit (Dyslexia), Wiesbaden, 1887.

[2] Neurologisches Centralblatt, 1891, S. 130.

[3] Ophthalmiatrische Beiträge zur Diagnostik der Gehirnkrankheiten, Wiesbaden, 1884.

Whether amnesic color-blindness may occur without right hemianopsia has not yet been ascertained. If it be found in right-handed persons, and —where the lesion is a solitary one—only with lesion of the left hemisphere (producing right hemianopsia), the explanation of the circumstance may, Wilbrand thinks, depend on the association fibres of both color-vision centres passing close to the visual centre on the left side, on their way to the cortical speech-centre; so that a lesion between their point of junction and the latter centre would be capable of producing amnesic color-blindness.

*Visual hallucinations*[1] sometimes occur in cases of homonymous hemianopsia in the blind side of the field only, or may precede the hemianopsia. Some of these observed by patients were: a military officer in uniform, a boy, a pig eating oats, chairs and tables, a sheet of water, smoke, etc. Henschen is of opinion that hemiopic hallucinations occurring in focal brain-disease indicate the position of the lesion to be in the occipital lobe, and holds that they result from irritation of the centre for visual memory. They would not occur if that centre were destroyed; but the lesion, he believes, is likely to be situated not far from it, either in the cortical centre for vision or in the subcortical fibres. De Schweinitz, however, has published a case[2] in which there were homonymous hemiopic hallucinations in those sides of the fields, which later on became hemianopic, and the lesion was found to be a gumma at the base pressing on the right optic tract. The author implies that there was no disease of the occipital lobe, but the latter does not seem to have been carefully examined.

Hallucinations of vision occur, as we know, in connection with certain forms of insanity, in delirium tremens, and in acute fever; and the writer has seen a case of chronic simple glaucoma, without any brain-disease, in which there were marked, but not hemiopic, hallucinations,—brilliant flower-gardens for the most part; and a few other cases of glaucoma with this symptom are recorded. There may, therefore, occasionally be some danger of an error in diagnosis when the symptom appears in focal brain-disease; yet the presence of other signs of focal brain-disease, and the fact that the hallucinations are always in one or the other side of the binocular field, and are either accompanied or followed by blindness of the same side of the field, should prevent any serious difficulty of diagnosis from arising. But in this connection it must be mentioned, that homonymous hemiopic hallucinations persisting for years without hemianopsia have been observed by Peterson[3] in a case of chronic delusional insanity. Uniocular halluci-

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[1] The terms hallucination and illusion must not be confounded with each other. A hallucination is an entirely subjective sensation,—an image which originates in the visual centre and is projected into space,—while an illusion is a subjective transformation of an actual sensory impression, as a face seen in the fire, or the sense of relief in a stereoscopic picture.

[2] The New York Medical Journal, May 2, 1891.

[3] Ibidem, August 30, 1890.

nations—referred to one eye only, and not seen when that eye is closed, and to be well distinguished from the hemiopic form—have been described by several authors as occurring in the insane.

Having set forth the visual symptoms caused by lesion of the visual centre, and of the occipital lobe in general, the disturbances of sight resulting from focal disease in the middle and anterior portions of the visual path, so far as we are acquainted with that path, will now be considered.

*b.* Lesions of the Posterior Third of the Posterior Limb of the Internal Capsule (Charcot's Sensory Crossway).—These lesions have been held, and are still held, by distinguished authorities to cause homonymous hemianopsia, along with hemianæsthesia of the opposite side of the body, and apparently the anatomical data for this opinion are present.

It has been generally and very reasonably assumed that, inasmuch as anatomists trace fibres running from the external geniculate body into the anterior quadrigeminal body, the pulvinar, and the posterior third of the posterior limb of the internal capsule, these three latter regions lie in the visual path. But the careful clinico-pathological investigations of Henschen of Upsala and others lead to a different conclusion. For when it is shown, that serious lesions of these parts can be present without causing hemianopsia; or that if, when hemianopsia is present with these lesions, the external geniculate body, or the optic tract, is likewise interfered with, either by inclusion in the lesion or by pressure, the only inference to be drawn is, that the former regions do not lie in the path of vision. The writer of this chapter is primarily concerned with the practical and clinical aspects of the subjects to be discussed, and it is not for him to attempt to reconcile any apparent conflict between anatomy and physiology on the one hand, and clinical pathology on the other, but simply to take the findings of competent observers in the latter department as they are presented. No one has ever before attempted such systematic work in the field of cerebral clinico-pathology as has Henschen, and his work bears the stamp of thoroughness in its methods and of soundness in its deductions, any adequate idea of which, in the space allotted to this chapter, it would be impossible to convey, and that work must, for the present, be accepted as the leading authority for clinicists on the many points of vast interest of which it treats. To the future we must leave the task of telling how the difficult physiological questions raised by it are to be answered; and in this connection the reader may be reminded, that the clinic and pathology have often held the lamp to cerebral physiology and anatomy.

Henschen points out,[1] that the analysis of published clinical cases which bear on the point renders little support to the commonly accepted theory, that a lesion confined to the posterior third of the posterior limb of the internal capsule gives rise to hemianopsia, or, in other words, that the

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[1] Brain, Spring Number, 1893, p. 175.

visual path passes this way. The writer, too, has been unable to find the record of any modern case with autopsy in support of the theory. In a case of Henschen's,[1] although the posterior limb of the internal capsule was completely destroyed, there was no hemianopsia. And in another case, in which a hemorrhage occurred in the internal capsule, hemianopsia at first was present, but ultimately passed away, while marked secondary degenerative changes, as displayed by the autopsy, remained in the capsule; temporary pressure on the external geniculate body, and not the lesion in the capsule, having obviously been the cause of the hemianopsia. In yet another case, a hemorrhage occupying the internal capsule, the thalamus, and pulvinar caused hemianopsia, but undoubted evidences of pressure on the external geniculate body were found. Yet another quite similar case was observed. Again, the posterior third of the internal capsule was found to be destroyed by a lesion of old standing, along with the whole of the pulvinar, without hemianopsia having been present during life.

The questions naturally arise, How does the visual path reach the external geniculate body from the optic radiations, if not through the posterior part of the internal capsule? and, What are these fibres which pass from the external geniculate body through the internal capsule to the optic radiations, if they be not visual fibres? The answers to these questions must for the present remain in abeyance, for want of definite information on the subject; but it must be remembered that some bundles of fibres have been described as branching off from the external geniculate body, and passing backward for a short distance in the outermost part of the crus (Wernicke), where they join the optic radiations of the occipital lobe as a direct root of the optic tract. It may be that these fibres, either wholly or in part, represent the visual path. At the same time, proof of such a view is also wanting, and Gowers thinks it doubtful whether any fibres have this direct path.

Professor Henschen does not speculate in his work as to an alternative path, but, from correspondence with which he has favored the writer, the latter learns that he inclines to the view, that the visual path passes directly from the external geniculate body into the optic radiations in the parietal and occipital lobes.

As regards the nature of the fibres which pass from the external geniculate body through the internal capsule and into the optic radiations, if Henschen be right in the distinction he makes[2] between visual and optic fibres in the optic radiations, the latter fibres being considered as reflex fibres, it may be that to this category of optic or reflex fibres alone belong those which pass through the posterior part of the internal capsule.

Charcot put forward the view,[3] that the symptom to which lesions of

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[1] Opere citato, iii. 88.

[2] Brain, Spring Number, 1898, p. 173.

[3] Leçons sur les localisations dans les maladies du cerveau, p. 114.

the posterior third of the posterior limb of the internal capsule give rise is crossed amblyopia,—that is, marked defect of vision, with very contracted field, in the eye away from the lesion, while the field of the eye on the side of the lesion is also contracted in a less degree. This view was supported by only a very few unsatisfactory post-mortem examinations. It led to the hypothesis of a double crossing of the optic fibres, and to the assumption of two distinct cortical centres, one for bilateral vision, the other for monolateral vision.

A few writers, most notably Gowers,[1] continue to maintain the theory of crossed amblyopia, although they assign the angular gyrus, and not the internal capsule, as the seat in which lesions causing this symptom are to be found. Ferrier's experiments, indeed, lend color to the view, that such lesions in animals do cause temporary blindness of the eye on the opposite side from the injured angular gyrus. But Gowers himself admits that there are "but meagre grounds for the assertion" that any such arrangement exists in man. It is remarkable that Gowers, with his abundant opportunities, is able to bring forward only one case with post-mortem examination from his own experience in support of the view he advocates, and can quote but two from the experience of others. The elaborate theory, which is built up on these meagre grounds, is as follows: In the angular gyrus there is a higher visual centre in which the half-fields are combined, and the whole opposite field is represented. The field of the same side is also represented in this centre, as shown by the fact that the amblyopia in the opposite eye is usually accompanied by a slight restriction of the field on the same side. Thus this centre is assumed to represent the whole of both fields, but that of the opposite side in a far higher degree than the field on the same side. This assumption is employed to explain the curious fact that the crossed amblyopia generally lessens after a time; for if the higher centre in each hemisphere is connected with both retinæ, the loss caused by disease of one hemisphere may, conceivably, be compensated by the function of the other hemisphere. Partial disease lowers the function of the centre as a whole, but does not, it is said, cause partial loss (hemianopsia) of one part of the field. Complete destruction of the centre does not cause complete loss of sight of the opposite eye; some vision and a small field remain, and these, it is believed, are due to the representation of this eye in the other hemisphere.

The vast majority of observers hold that the so-called crossed amblyopia has no existence, and there can be but little doubt that in those few cases where it has been supposed to be present an error of observation has been committed. This may have arisen, for example, in one of the following ways: either the true nature of the defect in the fields was homonymous

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[1] Manual of Diseases of the Nervous System, 2d ed., vol. ii. pp. 22 and 159, and Bowman Lecture, Transactions of the Ophthalmological Society of the United Kingdom, vol. xii.

lateral hemianopsia with marked peripheral contraction of the seeing side of the fields (see also p. 551) (that the eye away from the lesion in cases of homonymous hemianopsia should sometimes seem to have a more contracted field than its fellow is probably due to the fact, that the nasal side of the field is normally the smaller side, and that there should be hemianopsia with lesion of the angular gyrus is, of course, perfectly natural, in view of the proximity of the optic radiations in the underlying white substance); or, the contraction of the fields has been purely functional in, say, a case of hysteria, which has become complicated, and perhaps aggravated, by the addition of organic brain-disease; or, the perimeter chart has been taken by an inexperienced assistant, for there is no method of functional examination with which erroneous results are more liable to be obtained than with perimetery, until the examiner has gained some experience.

*c.* LESIONS OF THE PRIMARY OPTIC GANGLIA (OPTIC THALAMUS, ANTERIOR CORPUS QUADRIGEMINUM, AND EXTERNAL GENICULATE BODY).—When both eyes are removed in a new-born animal, the optic nerves degenerate, and a large part of both optic tracts undergoes atrophy, as do also the external geniculate bodies, the anterior corpora quadrigemina, and the pulvinares. In lesions of the occipital lobe secondary degeneration has been traced to, and found in, these ganglia. Hence it is proved that there is, indeed, a relation between these ganglia and certain fibres passing from the occipital lobe to the optic nerve, and *vice versâ;* the nerve-fibres, it is found, do not pass through the ganglia, but end or begin there in the processes of the ganglionic cells. But when we come to look for clinical evidence of the passage of the sensory path through the primary optic ganglia, we are at fault. This evidence has been hitherto indefinite and conflicting, but Henschen's views on the subject must now prevail.

*Optic Thalamus.*—It is into the pulvinar, or posterior part of this ganglion, that fibres from the optic tract and optic radiations respectively pass.

Lesions of the pulvinar are often accompanied by hemianopsia, but the autopsies have usually shown that the lesion extended beyond the pulvinar, and involved the visual path outside it. In a case of Henschen's, a hemorrhage bulged out the pulvinar posteriorly, so that the visual path may readily have been pressed up; while in a second case the pulvinar was destroyed, except its ventral portion, but hemianopsia was not present. In a third case, already mentioned, the pulvinar was wholly destroyed, along with the posterior third of the internal capsule, without hemianopsia. In yet another case permanent changes in the pulvinar were produced by a hemorrhage, and without hemianopsia. Zacher's case,[1] also, shows that almost complete degeneration of the pulvinar is not accompanied by hemianopsia. Yet some of Henschen's cases seem to show that the pulvinar is necessary for sight, but they do not prove it. In nine cases collected by

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[1] Archiv für Psychiatrie und Nervenkrankheiten, xxii. S. 661.

Sinkler[1] there was no uniformity of symptoms, those most commonly observed being somnolence and impaired sensibility and paresis of the opposite side, the two latter being probably ascribable to the lesion of the internal capsule which was present in most cases, as was also lesion of the corpora quadrigemina. In a case of Dercum's,[2] with a tumor of the left optic thalamus involving chiefly the pulvinar, and extending to the neighboring part of the nucleus caudatus, there were athetosis, hemianop-ia, and the hemianopic pupil. The two latter symptoms are explained by the author as being due to pressure on the optic tract. In a case of Zenner's,[3] a tumor of the optic thalamus was accompanied by hemianopsia, but that the latter was a distant symptom, and not the immediate result of the lesion in the thalamus, is plain from the fact that the lesion and the hemianopsia were both left-sided. The author believes the hemianopsia to have been due to pressure on the optic tract by the distended third ventricle. Gowers[4] states that hemianopsia may be caused by disease limited to the posterior part of the thalamus, and that he has seen two cases in which there was no other lesion that could be associated with the hemianopsia, which existed during life.

It cannot be denied that lesions of the internal capsule and of the pulvinar are very frequently attended by hemianopsia, and hence—although this symptom here is a distant one, due to the great liability to pressure on the visual path (external geniculate body) which is so often present—it must, in a sense, be reckoned as a symptom of these lesions; but less so in cases of softening, where transmitted pressure is not present, than in hemorrhage and tumor. There are, indeed, two symptoms, which, when they occur together, are regarded as typical of a lesion in the pulvinar,—namely, athetosis (caused probably by irritation of the motor path in the internal capsule) and hemianopsia, and there is often, also, hemianæsthesia.

(*Corpus Striatum.*—It is convenient to mention here that Nothnagel has described[5] the following train of symptoms in lesions of the corpus striatum. 1. Signs of vaso-motor paralysis of the side of the body away from the lesion,—elevation of temperature, redness and œdema of the skin. 2. Apparent ptosis on the paralyzed side, owing to contraction of the palpebral aperture, but the lid can be raised. 3. A shrinking back of the eyeball into the orbit, so that it seems to have become smaller. 4. Contraction of the pupil of the same eye. 5. Abnormal secretion of thin mucus from the corresponding nostril, of tears from the affected eye, and of saliva from the corresponding side of the mouth. The writer is not acquainted with more recent observations of this kind.)

*Corpora Quadrigemina.*—What the symptoms are, which hemorrhages

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[1] The American Journal of Ophthalmology, December, 1893.
[2] The Journal of Nervous and Mental Diseases, 1890, p. 586.
[3] Neurologisches Centralblatt, 1893, S. 607.
[4] Diseases of the Nervous System, 2d ed., ii. p. 316.
[5] Loco citato, p. 327.

or softenings strictly limited to the corpora quadrigemina produce, is not yet known. Such lesions are rare, owing to the fact that these organs have their vascular supply from the posterior cerebral artery, in common with the crus cerebri, the posterior part of the lateral ventricle, the optic thalamus, and the occipital lobe. It is not long ago since it was strongly held that the corpora quadrigemina were intimately connected with sight. Griesinger stated, "If vision be intact, the seat of the tumor can never be in the corpora quadrigemina (the intra-cerebral seat of vision)." But in 1879, Nothnagel,[1] after a careful examination of the evidence to hand, arrived at the conclusion that a tumor might very well implicate the corpora quadrigemina without affecting vision, and the writer is not acquainted with a case of more recent date which points to any other conclusion. The following are the only two cases which lend color to the opposite view. In a case mentioned by Charlton Bastian,[2] and quoted by Nothnagel, a long illness was brought to a conclusion by blindness for about fourteen days, and a patch of softening was found almost limited to the anterior corpora quadrigemina. Eisenlohr has recorded[3] a case in which a revolver bullet lodged in the right anterior corpus quadrigeminum, and the author thinks that the blindness of the right eye, which came on five months later without optic neuritis, is to be regarded as the result of the lesion of the quadrigeminal body *per se*, or of the right colliculus anterior. But the fact that optic neuritis was found in the right eye two months subsequently, taken with the long interval between the injury and the onset of any defect of sight, seems to render the evidence afforded by the case against, rather than in favor of, the author's views. As opposed to these inconclusive positive cases, there are many negative ones, in which tumors of the corpora quadrigemina caused no blindness; while in others, where blindness was present, it was found, in the properly observed cases, to have been accounted for by the presence of optic neuritis. (Compare vol. i. p. 63 of this System.)

(The oculo-motor disturbances caused by tumors of the quadrigeminal region are treated of at page 597.)

*External Geniculate Body.*—This is the only one of the primary optic ganglia which Henschen and all other observers unhesitatingly connect with the act of vision. Into it pass all the fibres from the optic tract, and its lesion invariably gives rise to hemianopsia. Moreover, in Henschen's opinion, the dorsal portion of the external geniculate body innervates the upper half of each retina, and the ventral portion the lower half of each retina. In the case on which he bases this view, there were, in the dorsal and dorso-medial portion of the right external geniculate body, marked disease-changes, which were associated with loss of the left lower quadrant in each field. Hence, quadrant hemianopsia may be caused by a lesion of the external geniculate body.

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[1] Topische Diagnostik der Gehirnkrankheiten.

[2] On Paralysis from Brain-Disease, p. 115

[3] Neurologisches Centralblatt, 1890, S. 747.

A more recent case[1] bears out this view. The patient, who was under the clinical care of Wilbrand, was attacked with apoplexy, and left hemiplegia and anæsthesia. At first there was complete left hemianopsia with concentric contraction of the right sides of the fields. Subsequently this resolved itself into left lower quadrant hemianopsia alone, which continued until the patient's death, more than three years later. To account for this, Henschen found at the post-mortem a hemorrhagic cyst, which had destroyed the dorsal half of the right external geniculate body, without injury to any other part of the visual path.

In an eight-months fœtus Flechsig[2] found myelization of the optic nerve in those fibres alone which correspond with the macula lutea, and he was able to trace these fibres into the external geniculate body. He holds that, if this observation be generally applicable, the macula lutea only, and no other part of the retina, stands in relation to the external geniculate body. It is difficult to see how this view can be maintained, at least for the mature optic nerve, when it is admitted that lesions of the external geniculate body produce hemianopsia.

*d.* Lesions of the Optic Tract (*d* and *e* should be read in connection with pp. 602 to 610).—The chief symptom of these lesions is homonymous lateral hemianopsia. The diagnosis between cases of hemianopsia due to lesions of the tract, and those due to cortical and subcortical lesions is, as a rule, not difficult, for in the former the symptoms (word-blindness, mind-blindness, etc.) that are apt to attend upon central lesions will be absent, while, owing to the liability to implication of the trunks of the cranial nerves in lesions at the base of the brain, paralyses of these nerves, and especially of those supplying the orbital muscles, are very liable to be present. Yet the tract may be diseased without any cranial nerve being involved, and cortical hemianopsia due to a lesion which is confined to the centre for sight is unattended by other symptoms.

The hemianopic defect in tract lesions may be incomplete. Henschen[3] records a case of lesion of the right tract, caused by a tumor pressing from above and from the outside, which gave rise to a defect in the lower left quadrant of the fields only; and he thinks this case supports his view, and completes the evidence in its favor, that the visual fibres to the ventral retinal quadrant run ventrally in the entire visual path from cortex to retina. Quadrant hemianopsia is not, then, pathognomonic of a lesion in the occipital cortex, but may attend lesions of the optic radiations, of the external geniculate body, or of the optic tract.

There is sometimes a slight difference in the position and extent of the defect in the fields, especially if the lesion be in the tract near the chiasma, where a complete mixing of the crossed and uncrossed fibres has not yet

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[1] Neurologisches Centralblatt, March 1, 1898.
[2] Gehirn und Seele, SS. 53, 71.
[3] Loco citato, ii. p. 376.

come about. By incomplete destruction of the conduction through the tract, the defect in the half-fields may be only relative, and hemiachromatopsia may be produced without loss of the form- or light-sense. Extension of the disease from the tract to the chiasma causes, in some cases, a defect of the hitherto sound side of the field, or, should the crus cerebri become implicated, there will be hemiplegia of the same side as that of the hemianopic fields.

Ophthalmoscopic signs are not uncommon, although by no means constant, in cases of lesion of the tract, or of the chiasma. These signs are sometimes congestion papilla, or descending neuritis, in cases of tumor or meningitis, or optic atrophy coming on without any preceding inflammatory process, and after a greater or less interval of time. The optic atrophy is often more marked in one eye than in the other, and it often attacks only that part of the papilla which is on the side of the diseased optic tract. Optic atrophy, on the other hand, is a very rare appearance with hemianopsia due to central lesions.

Wernicke's pupil-symptom, or the hemianopic pupil, is an important aid in the diagnosis of lesions of the optic tract when it can be observed. It was inductively formulated by Wernicke in the year 1872,[1] but not actually observed until some years afterwards, and there are still those who dispute its occurrence. The symptom consists in this that, when light is thrown on the blind half of the retina, the reflex pupil-contraction does not take place, or is sluggish, but can be observed as a prompt reaction when the sound side is illuminated.

We are well acquainted with the efferent path for the pupillary light-reflex (third nerve nucleus, third nerve, short root of ciliary ganglion, ciliary ganglion, short ciliary nerves, sphincter pupillæ), but we are not so certain as to the whole course of its afferent path. It is held that, like the visual path, it passes up the optic nerves, and, decussating in the chiasma, passes along the optic tracts. It leaves the tracts at some point not yet ascertained, but probably very near the geniculate bodies, to go to the gray matter of the aqueduct of Sylvius. By some it is believed that, before branching off, this afferent path passes into the external geniculate bodies, or into the anterior corpora quadrigemina, or into both ; but the pupil may react after the removal of these ganglia.

Van Gehuchten[2] states he has been able to confirm the researches of Hans Held to the effect, that there exist in the nuclei of the anterior quadrigeminal bodies large cells, the axis-cylinder processes of which are directed forward and inward, and, passing in front of the nuclear origin of the third nerve, decussate in the raphe, and then bend downward to unite with the posterior longitudinal fasciculus. These fibres, he states, give off branches, which ramify in the nuclear origin of the third nerve, and, lower

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[1] Virchow's Archiv für pathologische Anatomie und Physiologie, lvi. S. 397.
[2] Anatomie du Système Nerveux, 1897, p. 686.

down, in the nuclear origins of the fourth and sixth nerves as well; thus establishing a morphological connection between the visual fibres of the optic nerve and all the motor nerves of the eyeball.

Henschen[1] inclines to think, that the centripetal pupil-fibres are to be found in connection with the mesial root of the optic tract, and he excludes altogether the external geniculate body from their path. They would appear, he says, to pierce the posterior section of the cerebral peduncle, and, with the other fibres of the mesial tract, to enter into relations rather with the internal geniculate body.

Hence, a lesion of the right tract, for example, will not only deprive the right half of each retina of its functions, and cause left homonymous hemianopsia, but will also interfere with the afferent path for the pupillary reflex in the tract, so that light falling on the right half of either retina alone will not cause any contraction of the pupil. But lesions beyond the right tract, in the right brain, which cause left homonymous hemianopsia, likewise deprive the right half of each retina of its functions, and yet, inasmuch as they do not interfere with this afferent pupil-reflex path, they do not give rise to the hemianopic pupil. It is obvious that lesions in each hemisphere, causing what is called double hemianopsia, as a result of which the patient might be completely amaurotic, would be unattended by loss of the pupillary light-reflex. The pupil there reacts to the stimulus of light, although the visual perception of light is lost.

The hemianopic pupil is sometimes not present, even when there is a lesion of the tract; probably because the special fibres for the pupil-reflex offer greater resistance to certain forms of lesion than do the visual fibres. It is also remarkable that occasionally, in one and the same case, the hemianopic pupil can be observed at one time, while at another, under the same conditions, it cannot be discovered. Here, probably, some nerve-filaments in the diseased tract are but partially destroyed, and can sometimes be stimulated so as to produce the pupillary action.

Again, in a case of Henschen's,[2] a tumor of the dura mater pressed against the fossa Sylvii, and, apparently by producing attacks of cerebral congestion, gave rise to transient hemianopsia which was accompanied by the hemiopic pupil.

It is conceivable, as Knies inductively shows,[3] that a lesion might be so situated as to interfere with the afferent pupil-reflex path between the tract and the third nerve nucleus on one side only, without there being any interruption in the course of the visual path, the result of which would be that on illumination of the different sides of the retina the hemianopic pupil reaction would be obtained without hemianopsia being present. As in uncomplicated cases the pupils are of equal size, and react equally to

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[1] Loco citato, Part III., p. 111, etc.

[2] Loco citato, ii. p. 870.

[3] Beziehungen des Sehorgans und einer Erkrankungen, etc., Wiesbaden, 1893, S. 116.

light in each eye, the condition will not be discovered unless it be specially searched for. Indeed, no case of hemianopic pupil-reaction without hemianopsia (Knies's pupil-symptom, as we may call it) has yet been recorded, although it is fair to assume that the symptom cannot be very rare.

It is necessary that great care should be exercised in looking for the hemianopic pupil, lest it should be thought to be present when not present, or be overlooked when really there. Even in healthy eyes, a variation in the angle of incidence of the rays of light passed into the eye from one or other side may produce a marked difference in the promptness of the pupil-reflex. It is important, therefore, that the intensity of the light, and its angle of incidence on each side of the retina, be as nearly as possible the same. It is usually a more sluggish reaction of the pupil, rather than complete absence of it, on illumination of the blind side of the retina which must be expected; for it is impossible to apply the test so that some light will not reach the good side of the retina, and from thence liberate a certain degree of pupil-reflex. A good method of applying the test is, one eye being closed, to throw the light from a concave ophthalmoscope mirror, by preference a short-focussed mirror, now on one side of the opposite retina and now on the other side, whilst a second observer notes any difference in reaction of the pupil which may exist. Or, perhaps, a better method is Schmidt-Rimpler's. One eye being closed, the patient is caused to direct his other eye to one side and to look at a distant object. A convex glass is then held before the eye, and light from the flame of a lamp is reflected through the glass from a concave ophthalmoscope mirror, so as to produce the smallest possible image of the flame on the retina. The pupillary reaction is then observed by throwing the light on and off the eye. The same process is repeated for the other side of the retina, and a comparison between the reflex which is released from each side of the retina is made.

It must not be forgotten that tumors of the crus cerebri, optic thalamus, lenticular nucleus, or temporo-sphenoidal lobe, by extension to, or by pressure on, the optic tract, may cause hemianopsia and the Wernicke pupil-symptom. A case published by Leyden[1] is of practical interest. The symptoms were left hemiplegia, left facial palsy, left ptosis, left homonymous hemianopsia, with the hemiopic pupil,—a group of symptoms which, on the whole, pointed to a lesion of the right optic tract and crus cerebri; but there was also conjugate deviation of the eyes to the right,—a symptom which pointed to a lesion in the right hemisphere. The autopsy discovered a tumor in the right lenticular nucleus, which extended into the crus cerebri and implicated the optic tract.

A case of Wernicke's[2] is important in the opposite sense. As the result of a stab in the head, the patient had homonymous hemianopsia and brachial monoplegia. The presence of the hemianopic pupil enabled the

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[1] Deutsche medicinische Wochenschrift 1892, Nr. 1.

[2] Allgemeine Wiener medizinische Zeitung, Nrs. 48 und 49, 1893.

diagnosis of a lesion of the crus cerebri with implication of the optic tract to be made with certainty.

Softening and hemorrhage rarely affect the tract. New growths, including tubercle, but, above all, syphilitic gummata and syphilitic meningitis, are the morbid processes which are chiefly found when the functions of the optic tract are interfered with.

*e.* Lesions of the Optic Chiasma.—The characteristic symptom of these lesions is bitemporal hemianopsia, by reason of implication of the decussating fibres at either the posterior or the anterior angle of the chiasma, or at both; as a result of which the functions of the median half of each retina are impaired. The alterations in the fields of vision very commonly begin by a peripheral contraction on the temporal sides of the field for colors only, soon followed by a similar contraction for the form- and light-senses, these all gradually advancing until complete bitemporal hemianopsia comes about; but a few cases are on record [1] in which, in one or both fields, there was for a time complete temporal hemiachromatopsia alone, or with loss of the form-sense but retention of the light-sense.

It seems that occasionally a lesion of the chiasma in its early stages may give rise to a central scotoma of an atypical form, and not to hemianopsia, which, however, comes on later. Care in the diagnosis in these cases is obviously required, lest they be mistaken for toxic amblyopia. Although some reference was made to such cases by Förster [2] and by Treitel,[3] yet to Nettleship [4] is due the credit of having definitely drawn attention to them. He records seven instances, in one of which a post-mortem was obtained. The cases, in the later periods, were attended by other head symptoms in the form of mental failure, frontal headache, and varying paralyses of one or other of the oculo-motor nerves. Nettleship believes it is disease which begins towards the anterior angle of the chiasma where the macular fibres lie, that makes itself known at first by a central defect in the field.

Unlike the majority of cases of homonymous lateral hemianopsia, bitemporal hemianopsia is almost invariably accompanied, even in its incipient stages, with diminution of the central acuteness of vision. In rare cases, when the process has destroyed the decussating fibres it comes to a stand-still, and bitemporal hemianopsia continues for the remainder of life as a permanent symptom. These are mostly cases of circumscribed meningitis, of periostitis, or of hyperostoses starting from the base of the skull, the progress of which has been arrested; but in the majority of cases of chiasmal lesion the uncrossed fibres subsequently become diseased, and complete amaurosis is the final result. Sometimes complete blindness of one

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[1] Berry, The Ophthalmic Review, iii. p. 165; Hill Griffith, The Medical Chronicle, January, 1887.

[2] Graefe-Saemisch, Handbuch der gesammten Augenheilkunde, 1877, vii. S. 116.

[3] Archiv für Augenheilkunde, xxv., 3, S. 68.

[4] Transactions of the Ophthalmological Society of the United Kingdom, 1897, xvii. p. 277.

eye comes on, before the nasal side of the field of the other eye has become affected.

The temporal field of each eye may be attacked simultaneously, or that of one eye may become defective a short time before its fellow. At first the ophthalmoscopic appearances are negative, but at a later period simple atrophy of the optic nerves nearly always develops, and often makes its first appearance on the median side of the papilla. Papillitis, or optic neuritis, even when the lesion is a tumor, is not often seen.

A case reported by Weir Mitchell[1] demands special mention, from its uniqueness. In it there was bitemporal hemianopsia, with central vision in each eye of $\frac{6}{60}$, while the motions of the orbital muscles and of the iris were normal, and there was no loss of smell. There was atrophy of the nasal side of each optic papilla, but no optic neuritis. At the autopsy a tumor the size of an orange was found to have produced absorption of the sella turcica, and to have burst asunder the chiasma in the middle line, each tract being directly continued into the optic nerve of its own side. The tumor turned out to be an aneurism originating in an anomalous artery, which had run underneath the chiasma, and had united the two internal carotids.

It has been pointed out by Oppenheim,[2] that transitory recurrent bitemporal hemianopsia may be caused by syphilitic tumors implicating the chiasma, and he regards this form of transitory hemianopsia as an important sign of basal syphilitic lesion. The fact, indeed, must be recognized, that the symptoms caused by syphilitic gummata at the base of the brain —their favorite seats being the chiasma and the interpeduncular space— are frequently inconstant: a nerve which is paralyzed to-day may be found to perform its function well to-morrow, while the paralysis of some other nerve may continue. At post-mortem examinations, too, it is often seen that nerves, from which no symptoms have been derived during life, are completely embedded in the gumma.

Bitemporal hemianopsia is much less frequent than homonymous lateral hemianopsia. To judge from the recorded cases, it probably does not form more than five per cent. of all cases of hemianopsia. Yet it may possibly be often overlooked, for the nature of the binocular field here is such, that many patients would not complain of blindness towards one side or the other, while the other symptoms—amblyopia, ophthalmoscopic changes, etc. —might be sufficient to engage the whole attention of those practitioners, who are not very systematic in their functional investigations.

If the chiasma be attacked at either of its lateral angles, instead of at its posterior or its anterior angle, the visual defect will be nasal hemianopsia of the eye on the corresponding side, by reason of the injury to the uncrossed fasciculus, and consequent loss of function in the outer half of the retina

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[1] The Journal of Nervous and Mental Disease, 1889, xiv. p. 44.

[2] Berliner klinische Wochenschrift, 1888, S. 584.

on this side, while the field of vision of the other eye will remain intact; but nasal hemianopsia is very rare indeed. Henschen's investigations[1] induce him to think that a tumor in the external angle of the optic commissure is apt to affect not only the uncrossed, but also the crossed fasciculi, and to produce a form of bilateral homonymous hemianopsia.

Horizontal hemianopsia, whether superior or inferior, may be caused by a growth, or an exudation, which presses on the chiasma from above or below (Henschen). Horizontal hemianopsia may also be caused, probably by symmetrical lesions in the hemispheres, and, we know, by optic neuritis.

In lesions of the chiasma, concomitant symptoms sometimes present are: anosmia and paralysis of the orbital nerves from pressure on the first nerve, and on the third, fourth, and sixth nerves in the cavernous groove; also anæsthesia of the conjunctiva and cornea, from pressure on the fifth nerve.

The lesions found in this region are very various: fractures of the body of the sphenoid, cysts, tubercular nodules, tumors (including those which may spring from the under surface of the frontal lobes), exostoses, meningitis (chiefly of syphilitic origin), internal hydrocephalus (the distended infundibulum of the third ventricle pressing on the middle of the chiasma), tumors of the cerebellum, which by closure of the aqueduct of Sylvius may cause internal hydrocephalus, and produce blindness in this way, periostitis, and, very commonly, syphilitic gumma. This last morbid growth may give rise to anomalous arrangements of the symptoms, owing to the erratic manner of its growth and pressure. In a case of Henschen's there was nasal hemianopsia of the right eye, with total blindness of the left eye, yet the whole chiasma was found embedded in a gummatous meningeal exudation.

But perhaps the most frequent cause of injury to the chiasma is tumor of the pituitary body, producing bitemporal hemianopsia. Proptosis of one or both eyes from encroachment of the new growth into the orbits, with loss of power of motion of the eyeball, has been seen in some of these cases; also, the discharge of muco-purulent or of muco-sanguineous fluid from the nostrils, loss of smell from involvement of one or both olfactory tracts, and often, among the general symptoms, drowsiness, excessive development of the subcutaneous fat, thirst, and diabetes mellitus or insipidus. Psammoma, which has its seat of election in the pituitary body, is a form of new growth which may often be found in these cases. Dimness of vision was the first symptom complained of in a case of that kind recorded by Woollcombe.[2] At the beginning, although complete blindness soon came on, there were no diseased appearances in the optic papilla; but subsequently this became atrophied. No trace of the optic commissure could be found at the autopsy.

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[1] Loco citato, ii. p. 238.

[2] British Medical Journal, June 28, 1894.

In that rare and remarkable disease, for a knowledge of which we owe so much to Pierre Marie, and to which he has given the name acromegaly,[1] bitemporal hemianopsia is one of the commonest and earliest symptoms, often, indeed, preceding the other symptoms for a long time,—in one case for five years,—while complete blindness is very usual in the later stages. This defective vision is due to simple hypertrophy of the pituitary body causing pressure on the chiasma. In addition to the prognathism in the lower jaw, and the enlargement of the extremities present in this disease, there are constantly present, Marie states, hypertrophy of the pituitary body, with dilatation of the sella turcica, persistence of the thymus gland, and hypertrophy of the cord and of the ganglia of the sympathetic system.

He maintains that autopsies in which these lesions were not found were not of patients suffering from true acromegaly.[2] But cases are on record in which there was no disturbance of vision. In such cases, if Marie be right in asserting the constancy of the hypertrophy of the pituitary body, it must be assumed that that hypertrophy did not attain dimensions sufficient to affect the chiasma. The hypertrophy of the pituitary body is usually looked on as one of the results of a common cause, not itself as the cause of the disease; but Arnold, in a recent and elaborate paper,[3] advocates the opposite view. Marie states that he has seen double optic neuritis in these cases; but its presence is certainly not the rule.

In advanced stages optic atrophy is present and the eyes sometimes become prominent.

In the skeleton of Magrath, the Irish giant (A.D. 1760), preserved in the Anthropological Museum of Trinity College, Dublin, and in whom during life, as the distinguished Professor of Anatomy in Trinity College, Dr. D. J. Cunningham, F.R.S., has pointed out,[4] acromegaly was undoubtedly present, there is abundant evidence that the optic commissure, and at least the right optic tract, must have been subjected to great pressure, and that there was exophthalmos of the right eye. The sella turcica is enormously dilated, the optic groove and olivary process in front have quite disappeared, and the right sphenoidal fissure is much enlarged, and through it, no doubt, the enlarged pituitary body encroached upon the orbit. To the writer it seems certain that the giant must at the least have had bitemporal hemianopsia. But it is probable that he was quite blind of his right protruded eye, and that he had very defective sight in the left eye, if he were not quite blind of it too. Yet in such accounts of the giant's life as are extant no mention of any defect of sight is made.

Uhthoff[5] has observed a unique case in which, with arrest of development of the skeleton at the ninth year, and marked reduction in size of the

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[1] Brain, July, 1889.

[2] See also P. Marie and C. Marinesco, Archives de médecine, Juillet, 1891.

[3] Centralblatt für allgemeine Patholog e und pathologische Anatomie, Juni 1894.

[4] The Transactions of the Royal Irish Academy, 1891, p. 553.

[5] Berliner klinische Wochenschrift, 1897, Nr. 22.

thymus gland, yet no myxœdema, but rather emaciation of the soft parts, there was temporal hemianopsia with ophthalmoscopic signs of atrophy of the optic nerves. The author believes that there was some diseased process at or in the neighborhood of the pituitary body.

Most authors are silent as to the presence or absence of the hemianopic pupil in cases of chiasma lesion, but Seguin observed it[1] in several such cases; it was present in a case of Dulles,[2] and the writer has now under his care a young lady with bitemporal hemianopsia and the hemianopic pupil.

As syphilitic gumma is so frequently the cause of damage to the optic tract and to the chiasma, it must be stated that in these cases, even when absolute blindness has come on,—provided it has not lasted long and that active antisyphilitic measures are employed,—a cure may often be effected to such an extent as to restore useful or even, in some cases, full vision, and this[3] although the optic papilla may have already begun to get pale.

*f.* Lesions of the Intra-Cranial Portion of the Optic Nerve.—Cases of disease of the intra-cranial portion alone of both optic nerves must be of extreme rarity, and attention will here be directed chiefly to monocular cases. Monocular blindness, either without ophthalmoscopic changes or with optic neuritis or optic atrophy, and, it may be, if the amaurosis be complete with loss of the pupil-reflex to light, are the main signs which would suggest the presence of this lesion. But these symptoms alone will be insufficient to enable a diagnosis to be made, with certainty, between disease of the intra-cranial portion of the nerve and disease of the intra-orbital portion.

Diseases, not merely of the optic nerve itself, but also inflammatory exudations and tumors which invade it in its intra-cranial course, may often be diagnosed by the monocular blindness (with or without optic neuritis, or choked disk, or optic atrophy), and implication of the orbital nerves without exophthalmos. If exophthalmos be present, the diagnosis of intra-orbital disease would prevail, unless where other symptoms pointed to thrombosis of the cavernous sinus (page 605), to aneurism of the internal carotid or of the ophthalmic artery (page 606), or to an intra-cranial tumor encroaching on the orbit. But in cases of monocular amaurosis without ophthalmoscopic changes, the question between hysteria and intra-cranial lesion of the optic nerve may be the one to be decided, and a lengthened observation of the cases can alone provide the desired certainty of diagnosis in many instances. Yet, where there is organic disease, the pupil of the affected eye will usually be permanently dilated and will not react to light, while in hysteria the size of the pupil and its reaction will be normal or variable. In the organic cases the amaurosis, if blindness have gone so far, will be absolute and constant, while in the hysterical cases small print will

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[1] The Journal of Nervous and Mental Disease, November, 1887.

[2] The Medical News, Philadelphia, November 5, 1892.

[3] Uhthoff, Archiv für Augenheilkunde, xl., 1, S. 237.

be read in the stereoscope. The diagnosis of hysteria sometimes arises, too, in cases of binocular amaurosis. Hysterical amaurosis commonly comes on as the result of a mental shock, and both eyes lose their sight at the same time and suddenly, and as suddenly regain sight; while amaurosis due to organic lesion of both optic nerves is very rare, and never sudden, and more centrally situated organic disease causing binocular amaurosis would probably be attended by some of the concomitant symptoms, which have been set forth in foregoing pages. Sudden or rapid binocular blindness due to toxic influences (as in diabetes) acting on the visual centres must be borne in mind in this connection.

Leber[1] inclines to think that many cases of monocular hysterical amaurosis, or amblyopia, and possibly some binocular cases, are not merely functional, but are really due to transitory inflammatory processes in the optic nerve, situated probably—as one post-mortem showed—close up to the optic commissure. In these cases the ophthalmoscopic appearances may be healthy, or optic neuritis may be evidenced by slight woolliness of the margins of the papilla; or, more commonly, slight pallor of the papilla, especially on its outer side, is found. It is open to question whether the cases indicated by Leber are not cases of disseminated sclerosis in an early stage.

In fractures of the base of the skull which pass through the optic foramen, the optic nerve at this point is very liable to become injured. It is torn or compressed, or hemorrhage into its sheath takes place. Blindness, either complete or partial, is present from the first, but it is only after a time that optic atrophy can be recognized with the ophthalmoscope. The fractures of the base of the skull which pass through the optic foramen are relatively frequent. Thus, Hölder[2] found that in eighty-eight fractures of the base the optic foramen was involved in fifty-four.[3] Blows on the forehead, or supra-orbital margin, are sometimes followed by amaurosis, which used to be referred to injury of the supra-orbital division of the fifth nerve. We now know that the lesion is organic damage to the optic nerve at the optic foramen, by reason of the concussion transmitted along the roof of the orbit, which causes fracture of the bone, or rupture of membranes or of blood-vessels. But falls and severe blows on the head, apparently without fracture of the base, are sometimes followed by blindness, and at a later period the optic nerve becomes atrophied. Probably in these cases the optic nerve has been momentarily compressed and contused in the optic foramen, in consequence of the concussion. Transient blindness following head injuries is often caused by hemorrhage into the sheath of the optic nerve.

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[1] Deutsche medicinische Wochenschrift, 1892, Nr 33.

[2] Bericht der ophthalmologischen Gesellschaft zu Heidelberg, 1874, S. 16.

[3] See also an admirable paper by S. Snell, Transactions of the Ophthalmological Society of the United Kingdom, xvii., 1897, p. 81, and Leber, Archiv für Augenheilkunde, xxvii., 1, S. 272.

Again, there are other rare cases of extravasation of blood at the base of the brain which either do not end fatally forthwith, as in a case of aneurism of the internal carotid reported by B. Bramwell,[1] or may even recover, as in a case of hæmophilia reported by the same author,[2] and in which the blood may find its way into the inter-vaginal space of the optic nerve, or commonly of each optic nerve, and give rise to optic neuritis discernible with the ophthalmoscope.

**B. Localizing Derangements of the Motions of the Eyeballs due to Focal Cerebral Disease.**—The cerebral lesions which cause derangements of the motions of the eyeballs may be divided into four groups, according to the situation they occupy,—namely: 1. Central lesions,—lesions situated in the cortex cerebri, or between the latter and the nuclei of the ocular nerves. 2. Nuclear lesions,—lesions which attack the nuclei of the ocular nerves in the gray matter around the aqueduct of Sylvius, and in the floor of the fourth ventricle. 3. Fascicular or radicular lesions,—lesions which attack the efferent fibres, or roots, of the ocular nerves in the crus cerebri or pons, after they leave their nuclei, and before they appear on the base of the brain at their apparent origins. 4. Basal lesions,—lesions which attack the trunks of the nerves at the base of the brain, between the pons and the sphenoidal fissure.

1. Derangements of Motion of the Eyeballs due to Central Lesions.—All motions of the eyeball which are innervated from the cerebral cortex are binocular, and associate, or, as they are more commonly termed, conjugate,—*i.e.*, both eyes simultaneously and with the like excursiveness look up, down, to the right, left, etc.; hence central lesions must cause derangements of conjugate movements of the eyeballs, and not of isolated eyeball muscles. The levator palpebræ is not included in this statement, for isolated central ptosis is a recognized symptom (*vide infra*).

*Conjugate Deviation.*—The disturbance of the mobility of the eyeballs due to central lesions is known clinically as conjugate deviation (often accompanied, in the case of conjugate lateral deviation, by a turning of the head in the same direction), the importance of which as a symptom attendant upon an apoplectic attack was first pointed out by Prévost.[3] It is also occasionally seen in cases of tumors and other diseased states. Inasmuch as the normal conjugate motion of the eyes, say to the right, is caused by a nervous impulse originating in the left hemisphere, a destructive lesion in the left hemisphere paralyzes the power of motion of the eyes towards the right, and consequently, by giving a preponderance to the forces which produce conjugate motion in the opposite direction, it produces a deviation of the eyes to the left, or, as Prévost graphically expresses it,

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[1] The Edinburgh Medical Journal, 1886, p. 97.

[2] Ibidem, p. 102. See also his Intra-Cranial Tumors, pp. 60, 62.

[3] De la déviation conjugée des yeux, etc., Thèse de Paris, 1868.

the eyes look at the lesion and away from the paralyzed side of the body. In irritative cerebral lesions or diseases, on the other hand,—*e.g.*, in Jacksonian epilepsy,—the eyes are turned away from the lesion and towards the convulsed side of the body. The above term—conjugate deviation—is reserved for defective lateral mobility due to central lesions; while the term conjugate paralysis (page 593) is applied to derangements of lateral motion due to certain of the second of the above-named classes of lesions,—nuclear lesions.

Conjugate deviation is usually a fleeting symptom. It passes away, as a rule, within a few hours after the onset of the apoplectic attack. The reasons for the evanescence of the symptom are: first, that it is most commonly a distant symptom; and, secondly, that—as with the innervation of groups of muscles elsewhere in the system, which work together symmetrically on each side of the body—the conjugate motions of the eyeballs are probably represented in each hemisphere, so that the sound side of the brain soon learns to perform the duty of the injured side.

The employment of conjugate deviation as a localizing symptom would be rendered difficult by the ephemeral nature of the symptom, were there no other obstacle; but a greater difficulty is due to the uncertainty which exists as to the position and nature of the cortical centre for these motions. Landouzy,[1] Wernicke,[2] and Henschen[3] on the evidence of clinical cases with post-mortem examinations, and Munk from experiments, localized this centre in the inferior parietal lobule. Ferrier found that electrical stimulation of the angular gyrus gave rise to conjugate deviation; but, as he held this region to be the centre for vision, he concluded that the reactions obtained were of the character of reflex movements set going by an impulse carried from the visual centre to the oculo-motor centre. He[4] localized this latter centre in the second frontal convolution, as did also Horsley and Beevor.[5] More recently, Schäfer[6] and Munk[7] have found that faradization of one visual centre (occipital lobe) caused associated motions of the eyes towards the opposite side, and that the eyes were turned downward when the stimulation was applied to the anterior zone of the centre and upward when applied to the posterior zone; also that stimulation of a certain region in the cortex, presumably the macular centre, caused either no motion or only one of adduction; and Munk showed that these motions come about even when the association paths between the visual and what he regards as the oculo-motor centres are cut across. From this Munk concluded, that

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[1] Le Progrès médical, 1879, pp. 36–49.

[2] Lehrbuch der Gehirnkrankheiten, ii. S. 58, and Archiv für Psychiatrie, xx., 1, S. 243.

[3] Opere citato.

[4] Functions of the Brain, 2d ed., p. 393.

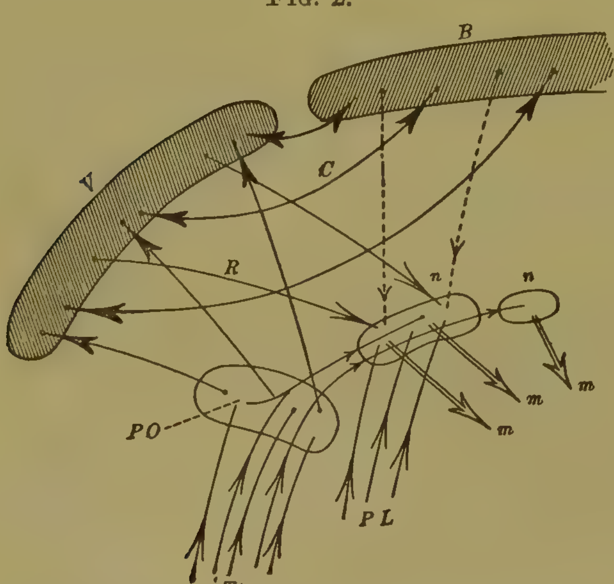
[5] Philosophical Transactions of the Royal Society of London, lxxix. p. 212.

[6] Brain, April Number, 1888.

[7] Brain, Spring Number, 1890.

involuntary fixation of the eyes on the stimulation of light is the result of a reflex from the visual centre through the optic radiations to the nerve nuclei. But Knies[1] puts forward the view that the cortical centre for vision is also the cortical centre for voluntary conjugate motions, so far as they are caused by conscious visual impressions; and points out that the motions obtained by Munk on stimulation of the visual centre represent voluntary and not involuntary motions. Not only are parts of the retina correlated with parts of the visual cortical centre in respect of sensory impressions, as is now usually held, but also, Knies believes, in respect of associated voluntary motions of the eyeballs. Hence, when a stimulus passes from the peripheral parts of the retinæ to the correlated parts of the visual centres, a voluntary impulse is transmitted directly from the latter to the nuclei in the floor of the fourth ventricle; with the result that certain associated motions of the eyeballs are thrown into action in such a way as to bring the maculæ luteæ and their cortical centres to bear on the object an image of which has been formed on the periphery of the retinæ. The more peripherally on the retinæ an image is formed, the more excursive must be the associated eyeball-motion in order to bring the image on the maculæ. By an effort of the will this motion can be inhibited. The macula lutea and its cortical centre are the only parts respectively endowed with the power of providing an accurate and brilliant mental image of objects. Motor pyramidal cells, it is stated, are contained in the visual cortex, and their axis-cylinders pass in the optic radiations to the oculo-motor nuclei in the pons. The path for these voluntary eyeball-motions due to visual stimuli, on this theory, would be such as is represented in the accompanying diagram (Fig. 2),—viz., *T, PO, R, V, R, n, m.*

FIG. 2.




*V*, cortical centre for vision: *B*, remainder of cerebral cortex; *R*, optic radiations; *C*, commissural fibres between the visual centre and other parts of the cortex; *PO*, primary optic ganglia; *T*, optic tract; *PL*, posterior longitudinal fasciculi; *n n*, nuclei in floor of fourth ventricle; *m m m*, motor nerves to eyeball muscles.

Voluntary motions of the eyeballs can, of course, be produced by other than visual impressions; most powerful of these are auditory and tactile stimuli. These would be brought about by aid of the path *B C V R n m*, or there may be a direct path, *B n*. This very possibly is the explanation of the fact, that faradization of the second frontal convolution in monkeys causes conjugate deviation, for it is close to the centre for the levator palpebræ, where

[1] Archiv für Augenheilkunde, xxiii., 1, S. 9.

also, it is reasonable to think, in analogy with what is held in respect of the Rolandic area, the centre for the cutaneous sensibility of the eyelids and for that of the surface of the eye resides.

In a case of Wernicke's and in some others, where a lesion of the inferior parietal lobule was the cause of conjugate deviation, Knies believes that the close neighborhood of this part to the visual cortex is sufficient to account for the production of this symptom.

Knies's view is largely borne out by the recent investigations of Flechsig.[1] According to this distinguished author, there is in the brain not merely one motor centre,—that one, namely, which governs the movements of the extremities, the face, and the tongue,—but at least two others: one in connection with the centre for hearing and the other in connection with the centre for sight. These secondary motor zones have no influence over the motions of the extremities, face, or tongue, but control the movements of the head and eyes. Flechsig traces the descending motor path from the visual sphere into the optic thalamus. From this ganglion he thinks it probable that there are reflex paths which conduct motor stimuli from the visual motor centre to other sensory motor centres,—*e.g.*, to the tactile centre (Rolandic area), where, as already stated, in the foot of the second frontal convolution, there is a region faradization of which causes conjugate motions of the eyeballs.

Altogether, it seems probable, that in an apoplectic seizure conjugate deviation comes about as the result of the suspension for the time of all the functions of the affected hemisphere, so that no stimuli can be transmitted to the visual centre, which itself is often paralyzed, as shown by the occurrence of transient hemianopsia (page 553).

Cortical blindness (*e.g.*, uræmic amaurosis) causes loss of the motions of the eyes in so far as these are the result of conscious visual impressions. In such cases the eyes can be voluntarily moved by stimuli received from other senses, especially hearing or touch, but these motions are merely approximate fixation motions; fine binocular fixation cannot be effected. In cases of hemianopsia, loss of motion is not so easily demonstrated; yet, as Knies believes, it is to some extent present. The eyes can follow an object towards the hemianopic side of the field, but the motions are awkward as compared with those towards the seeing side of the field. Knies thinks, too, that the difficulty in reading in right homonymous hemianopsia (page 554) is mainly due to some impairment of the voluntary conjugate motion of the eyes towards the right.

Nystagmus must be regarded, in many instances, as the manifestation of an imperfect control of the visual centre over the motions of the eyeballs, as, for example, where there is congenital amblyopia or amblyopia acquired soon after birth, the nystagmus being probably due to defective development of the paths between the primary optic ganglia and the visual cortex.

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[1] Loco citato.

The nystagmus which occurs in disseminated sclerosis is possibly caused by interference with the functions of the motor axis-cylinders from the visual cortex.

*Central Ptosis.*—There can be no doubt that a separate cortical centre for the nervous supply of the levator palpebræ and orbicularis palpebrarum exists, and this is not inconsistent with the fact that, as regards the motions of the eyeballs, associated centres alone are present; for although, as a rule, the elevators of the eyelids are associated in their motions, yet by an effort of the will most people can throw one of them into motion separately, or more than the other. Doubtless the power to innervate voluntarily one levator and orbicularis alone varies in different individuals, and Nothnagel leans to the opinion,[1] that in many persons the levator centres are practically associated centres, and that, consequently, central ptosis is rarer than it otherwise would be. The centre for the levator palpebræ is believed[2] to be in front of the upper extremity of the ascending frontal convolution, close to the arm centre; and, as in the motor zone for the extremities there is present the centre for sensation in those parts, so, probably, the centre for the levator palpebræ contains the cortical centre for that portion of the fifth nerve which supplies the surface of the eyeball. A destructive lesion of this centre causes ptosis of the opposite upper eyelid; and the conjugate motions of the eyeballs, which physiologists have obtained on stimulation of this same centre, may have been similar to the voluntary motions of the eyeball, which take place on irritation of the skin of the eyelids, or of the surface of the eye. Bruns,[3] Landouzy, and Wernicke believe this centre to be in the inferior parietal lobule. Central ptosis is not a very uncommon symptom, and it is probably often a distant symptom.

*The orbicular sign* is one which may be noticed in some attacks of apoplexy with hemiplegia, after consciousness has returned. It consists in this, that the hemiplegic person, who during health has been able to close each eye separately, and who even now can close both eyes together, or the eye on the sound side alone, is unable to close the eye on the paralyzed side by itself. This sign usually passes away after a short time. Sometimes with this sign it may be noticed that, when both eyes are closed, the eyelids on the paralyzed side are brought together with some effort, and that the other muscles of the upper part of the face supplied by the seventh nerve are somewhat wanting in power.

The writer observed this symptom very well marked and persistent in a case of Dr. Wallace Beatty's in which there was copious diabetes insipidus, but no apoplexy or hemiplegia. The diagnosis was obscure, but a gross cerebral lesion was suspected; the case, however, passed from under notice without further symptoms developing. The patient was very posi-

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[1] Topische Diagnostik der Gehirnkrankheiten, S. 454.

[2] Knies, Die Beziehungen des Sehorgans und seiner Erkrankungen, etc., SS. 75, 85.

[3] Neurologisches Centralblatt, 1898, S. 857.

tive in her statement, that previously she had been able to close each eye by itself and separately from the other, and this she could now do only with one eye. In each eye there was detachment of the retina, which had come on with the diabetes. In a case of hemiplegia, under the care of Sir C. Nixon, Dublin, Louis Werner observed the orbicular sign. The patient recovered sufficiently to leave hospital, and has not since been heard of.

*Oculo-Motor Disturbances due to Lesions in the Optic Radiations.*—These motor derangements are similar in character to those caused by cortical lesions, and rarely can be diagnosed from the latter, and then only, should they last long enough, by aid of the concomitant symptoms of an attending hemianopsia (no loss of pupil-reflex or of visual memory, while hallucinations and subjective sensations of light may be present, etc.). But somewhere in the neighborhood of the fourth ventricle the motor and sensory fibres must become separated, and a lesion here would interfere with the voluntary eyeball-motions without causing any disturbance of vision. Were the motor fibres from the macular cortex alone diseased at this place, the power of accurately converging the optic axes on the visual object by simultaneous innervation of the internal recti (fusion of the double images) would be affected, as Knies points out, while the other conjugate motions and vision would remain perfect. The imperfect fusion which sometimes occurs in tabes may be due to disease at this place.

2. Nuclear Ocular Palsy, or Ophthalmoplegia.—In this condition, the loss of power of the ocular muscles is the result of primary disease (degeneration, hemorrhage, softening, inflammation) of the orbital nerve nuclei in the central gray matter of the aqueduct of Sylvius, and floor of the fourth ventricle. Tumors in the aqueduct and fourth ventricle, or in their neighborhood (corpora quadrigemina, page 597), are capable of producing defective motions of the eyeballs similar to those of primary nuclear palsy, but these cases are not included in the term ophthalmoplegia, which it is desirable to retain exclusively for the primary disease. It must be admitted, that during life it may be impossible to make the diagnosis where only a very small tumor is concerned. Larger tumors of this region will probably be associated with the general cerebral symptoms belonging to intra-cranial tumors.

Primary nuclear paralysis may affect the exterior muscles of the eyeball only, and is then called exterior ophthalmoplegia; or it may affect the interior muscles only,—the sphincter iridis and ciliary muscle,—and is then called interior ophthalmoplegia; or both of these sets of muscles may be paralyzed, when the term mixed ophthalmoplegia is applied. The ophthalmoplegia may be complete or incomplete: the former, if all the muscles, interior (complete interior ophthalmoplegia) or exterior (complete exterior ophthalmoplegia), or both (complete mixed ophthalmoplegia), as the case happens, be affected, although perhaps not every muscle in an equal degree; the latter, if one or more of the muscles have quite escaped; consequently the combinations met with are numerous. One eye alone may be attacked

(complete, partial, or mixed monocular ophthalmoplegia), or both eyes may be paralyzed (complete, partial, or mixed double ophthalmoplegia). Probably the most common, and certainly the most characteristic, case is that of complete or incomplete double exterior ophthalmoplegia. In the complete cases the eyeballs are as immobile as though fixed in cement; in the incomplete cases those groups of muscles which are associated in their actions are the most liable to be attacked. The monocular cases are rare. The levator palpebræ is not often affected, and even in cases of complete exterior ophthalmoplegia, when ptosis is present, it is usually incomplete.

Nuclear palsy may be congenital and non-progressive. Congenital ptosis, which is frequently combined with loss of power in the superior rectus, and is usually binocular, is of this nature.

A peculiar congenital nuclear affection is described by Sydney Phillips.[1] When the patient looks to the right or to the left, the upper lid of the other side droops so as to produce nearly complete ptosis, while the upper lid on the side towards which the patient directs the eyes remains raised. Phillips suggests, as an explanation of these cases, an unusually close commissural connection between the nuclei of the two third nerves, which causes relaxation of the levator along with that of the internal rectus.

Another remarkable form of congenital nuclear palsy and spasm is that in which congenital monocular ptosis is associated with spasmodic movements of the affected eyelid during the action of certain muscles of the jaw. The first of these cases was reported by Marcus Gunn,[2] and since then some twenty similar cases have been recorded. In Gunn's case, with slight congenital ptosis and myosis on one side, the affected eyelid was raised quickly and powerfully when the external pterygoid of the same side was put in action, and this position of the eyelid was maintained as long as the jaw was kept drawn to the right. In eating, the effect was startling. A committee, of which Gowers was a member, examined the case, and reported that there must be an abnormal connection between the central mechanism for the external pterygoid and the levator muscle, the simplest explanation being that the levator is innervated both from the nucleus of the third nerve and from the external pterygoid portion of the fifth nerve. Since the innervation from the third nerve nucleus is defective, it may be assumed that in these cases some of the fibres of the levator palpebræ portion of the third nerve arise, not from the nucleus of the third, but from that of the motor part of the fifth. The condition always remains unchanged through life.

Acquired nuclear palsy is either chronic or acute, and of these the chronic form is much the more common.

Chronic nuclear palsy, called chronic poliencephalitis superior by Wer-

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[1] Transactions of the Ophthalmological Society of the United Kingdom, vii. p. 306. See also Browning, ibidem, x. p. 187.

[2] Transactions of the Ophthalmological Society of the United Kingdom, iii. p. 283.

nicke,[1] may exist at, or appear soon after, birth or in the early years of life, and may remain *in statu quo* all through life without becoming worse, and without becoming complicated with other nerve-lesions. But the disease in these infantile cases can be also for many years of the patient's life slowly progressive, until it finally becomes stationary. Interior ophthalmoplegia is rare here, but all six orbital muscles in each eye may be paralyzed, and ptosis is the rule. This progressive infantile form has a tendency to be accompanied by nuclear paralysis of the facial nerve leading to facial atrophy; and the ocular paralysis, once established, does not undergo cure. It does not seem that a complication with bulbar symptoms is to be feared.

It is more usual to meet with chronic nuclear palsy which has come on in adult life. Many of these cases deserve the term stationary, inasmuch as, a certain number of muscles having become paralyzed in a short time, the others remain healthy, and the condition becomes confirmed, no extension to the bulbar nuclei taking place.

But the most common form is what is known as chronic progressive ophthalmoplegia, appearing in adult life. In it the disease usually attacks the nuclei of only one or two muscles at first, and often those of associated muscles,—*e.g.*, those which move the eyes upward or downward or from side to side,—the power of convergence being maintained, and in its progress, likewise, it may be associated movements which successively become impeded. Moreover, starting at any part of the nuclear region, the disease may spread forward or backward. In this way it may not extend beyond the nuclei of the third and fourth nerves, or, beginning in them, it may extend forward to the nuclei for the interior muscles, or it may begin in the latter and extend backward. Again, beginning in the nuclei in the floor of the fourth ventricle, with some of the symptoms of bulbar paralysis, it may after a time attack those of the eyeball muscles, or the disease beginning in the ocular nuclei may spread to the bulbar nuclei.

Very occasionally, as in three cases observed by Hughlings Jackson,[2] in place of ptosis, there is a partial paralysis of the orbicularis palpebrarum, so that, in washing the face, soap gets into the eyes, and, even with the greatest effort at closure, light is still seen between the eyelids, and little resistance is offered to passive elevation of the upper lid. The probable explanation of this is, that the oculo-facial group of muscles (frontalis, corrugator supercilii, and orbicularis palpebrarum) is innervated from the third-nerve nucleus by way of the posterior longitudinal bundles, the fibres passing then in the trunk of the facial nerve to supply the group in question. The experiments of Mendel[3] render the existence of such an arrangement very probable, and it also receives support from a case of

[1] Lehrbuch der Gehirnkrankheiten, iii. S. 468.
[2] The Lancet, July 15, 1893.
[3] Neurologisches Centralblatt, 1887, S. 537.

bulbar paralysis which was carefully studied by Tooth and Turner,[1] and from three unique cases of isolated paralysis of the orbicularis of the upper lid observed by Silex.[2] Possibly this partial lagophthalmos might be found more often in case of ophthalmoplegia exterior, were it systematically sought for.

Ptosis is not very common in chronic progressive ophthalmoplegia, and the interior muscles frequently escape. The progress of the disease often covers a number of years. At the commencement diplopia is complained of, but after a time it very often disappears without any improvement in the ophthalmoplegia. The general health is for the most part satisfactory, although there is often a good deal of cerebral lethargy. But, if the disease spread to the bulbar nuclei, distressing symptoms are produced, and even typical bulbar palsy with a fatal result is sometimes seen. Moreover, in chronic nuclear palsy, the disease, by spreading to the ganglion cells of the anterior cornua of the spinal cord, may lead on to progressive muscular atrophy.

In the foregoing, chronic ophthalmoplegia has been represented either as occurring by itself, or as becoming complicated only with its prototypes, —bulbar paralysis, more or less well marked, and progressive muscular atrophy; but the disease sometimes complicates other forms of disease of the nervous system. *a.* Tabes. (See also page 637.) Diplopia due to paresis or even complete loss of power in one or two orbital muscles, which soon passes away to recur occasionally, is not an uncommon symptom in the prodromal stages of locomotor ataxy. Its cause is probably a vascular disturbance, or a slight ependymitis, in the nuclear region. On the other hand, the complete and permanent paralysis of an isolated orbital muscle, which is sometimes seen in tabes, is probably to be regarded as due to a peripheral, and not to a nuclear disease of the nerve. But well-marked nuclear paralysis, affecting a number of the muscles of each eye, does sometimes appear coincidently with the earlier symptoms of tabes, or it may precede them, or it may appear some months later. In these cases bulbar paralysis has sometimes also been noted. *b.* Disseminated sclerosis. (See also page 612.) The nuclear paralysis here is due to sclerosed patches in the nuclear region, which, according to their distribution, may give rise to any degree or kind of ophthalmoplegia, although the interior muscles are not often attacked. At first the paralyses are usually slight and temporary. Nystagmus is a symptom often seen in disseminated sclerosis, both with and without ophthalmoplegia; also twitchings of the eyeballs, especially at the extreme positions of their motions; but it is not yet known whether this symptom is to be referred to central disease, to nuclear lesions, or to peripheral affections of the muscular nerve-filaments.

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[1] Brain, Winter Number, 1891, p. 491; The Royal London Ophthalmic Hospital Reports, December, 1892.

[2] Archiv für Augenheilkunde, 1896, xxxii. S. 95; and *vide infra*, p. 672.

c. General paralysis (page 617). Nuclear ocular paralyses are occasionally observed in this connection. *d.* Exophthalmic goitre. Some cases of chronic ophthalmoplegia are recorded as occurring in this disease, and they lend support to the view of those who hold that Graves's disease is due to bulbar nuclear disease. If the levator palpebræ be paralyzed, the aspect of the patient will be markedly different from that which is usual in Graves's disease. But in this disease some care must be exercised in making the diagnosis of ocular palsy, lest defective motion, due merely to the proptosis, be mistaken for it. *e.* In severe cases of multiple neuritis ophthalmoplegia is occasionally seen, but it is probably even here sometimes due to nuclear disease, rather than to peripheral disease of the ocular nerves. Ophthalmoplegia, especially ophthalmoplegia interna, is also found as a post-diphtheritic affection, and more rarely after measles and scarlatina. In some of these cases the lesions are probably nuclear, but in others they may be regarded as of the nature of peripheral neuritis.

Of acute nuclear palsy there are two kinds: 1, the peracute or sudden nuclear palsy, and, 2, the subacute nuclear palsy.

The peracute, or sudden, nuclear palsy is the form first described by Wernicke,[1] under the name of acute hemorrhagic poliencephalitis superior, in contradistinction to acute poliencephalitis inferior, or acute bulbar paralysis. Since Wernicke's two first observed cases only some fourteen or fifteen others have been recorded,[2] and hence it would seem to be a rare diseased condition. Chronic alcoholism is its nearly constant cause.

The onset of the ophthalmoplegia is very rapid, or almost sudden, and is usually accompanied by much headache, somnolence, delirium, and vertigo; also by vomiting, painful cramps in the extremities, and unsteadiness of gait, or even marked loss of power in the legs. In two instances there was optic neuritis, and, although this was not of the congestion-papilla type, yet it is evident that when the symptoms are taken together, some of these cases may be mistaken for intra-cranial tumor. The temperature was above normal in one case only.

The ophthalmoplegia is liable to vary in its degree from day to day. The symptoms increase rapidly in violence, and the patient dies in from two to fifteen days, recovery having occurred in but one case. Death is caused by the supervention of acute bulbar paralysis, or by the alcoholic or other poisoning to which the condition is due.

The most constant and most marked post-mortem appearances in these cases are innumerable miliary hemorrhages in the central gray matter of the third and fourth ventricles and of the aqueduct of Sylvius. There are also signs of inflammation consisting in prolification of the ependyma, as evidenced by thickening with the formation of minute granulations. Occasionally, hemorrhages are seen throughout the whole of the mid-brain, in

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[1] Lehrbuch der Gehirnkrankheiten, ii. S. 229.

[2] See Boedeker's paper in Archiv für Psychiatrie und Nervenkrankheiten, 1895, xxvii S. 810, and Murawieff's paper in Neurologisches Centralblatt, Nrs. 2 und 3, 1897.

the white as well as in the gray substance. A diseased state of the coats of the vessels has been actually observed in but two or three of the cases; yet the hemorrhages, the engorgement of the vessels, and the abundance of very small vessels present, all indicate that there is vascular disease.

Inasmuch as direct lesion of the nuclei of the paralyzed orbital muscles by the hemorrhages is not always present, the ophthalmoplegia cannot be regarded as being caused by nuclear disease in the strict sense of the term, but is probably due either to functional derangement, the result of the proximity of the disease, or it is an effect of the general toxæmia on the nuclei.

The subacute nuclear palsy comes on less suddenly, and progresses more slowly. Its initial stage is often attended with headache and lethargy, but other head-symptoms are not marked. It is frequently uncomplicated by bulbar paralysis, and, even when so complicated, it is capable of terminating in partial or complete recovery within a few weeks or months.

*Etiology of Nuclear Palsy.*—The primary cases of this affection have as their most common causes syphilis and alcoholism. Those cases which occur in young children are possibly caused, in many instances, by a congenital predisposition, the result of the existence of one or other of these states in their forefathers. Other causes observed have been exposure to cold, poisoning by nicotine, lead, sulphuric acid, carbonic oxide, and tainted meat.

Nuclear palsy has been seen with diabetes and with purpura, and as a sequela of epidemic influenza (la grippe). Traumatic nuclear palsy may occur from blows or falls on the head without fracture of the skull, the form of lesion being hemorrhage in most instances, but sometimes the mere concussion appears to be competent to derange the nuclear functions. Paralyses of orbital nerves also occur as the result of fractures of the base of the skull (page 602), and it is not always possible where there are no concomitant symptoms to distinguish these cases from cases of traumatic nuclear paralysis. One case[1] is recorded in which both sixth nerves were paralyzed by a stroke of lightning, the lesion being probably nuclear. In a certain number of cases no cause can be assigned, and Bristowe[2] and others record cases of functional ophthalmoplegia.

*Pathology of Nuclear Palsy.*—In chronic progressive ophthalmoplegia, a simple atrophic degeneration of the cells of the nuclei, as was proved first by Hutchinson and Gowers,[3] and afterwards by Buzzard,[4] Ross,[5] Westphal, and Siemerling,[6] is the starting-point of the diseased condition; but the entire motor neuron (nucleus, nerve-trunk, muscle) often undergoes degener-

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[1] Emerson, The New York Medical Journal, 1886, pt. i. p. 520.

[2] Diseases of the Nervous System.

[3] Medico-Chirurgical Transactions, 1879, lxii. p. 312.

[4] Brain, v. p. 37.

[5] Ibidem, April, 1886, p. 24.

[6] Archiv für Psychiatrie und Nervenkrankheiten, xxii. und xxix.

ation. In the other forms the following changes have been found in the nuclei: hemorrhage, softening the result of thrombosis, acute hemorrhagic inflammation, and acute or chronic inflammatory degeneration. The degenerative changes are sometimes seen to extend to the nerve-roots.

*Treatment* must in the first instance be directed to the primary cause, if it can be ascertained, and, where the cause is unknown, strychnine and large doses of iodide of potassium are recommended.

*Diagnosis.*—The diagnostic signs of nuclear ocular palsy are: its usually bilateral occurrence, the frequency with which either the interior or the exterior ocular muscles alone are paralyzed, the frequently symmetrical affection of the muscles, and the frequent integrity of the elevators of the eyelids.

The third is by far the most common nerve to be attacked, either alone or with the sixth and fourth nerves; and the immunity of the levator palpebræ and of the interior muscles of the eye, so often observed with exterior ophthalmoplegia, can only be with a nuclear lesion; for, in the aqueduct of Sylvius the nuclei for the iris and ciliary muscle and for the levator lie at some distance from the nuclei for the other muscles supplied by the third nerve, while third-nerve paralysis from a lesion of the trunk of the nerve at the base of the brain is more likely to involve a l the branches of the nerve. But the levator and interior muscles may be attacked in nuclear paralysis, and in that case the bilateral nature of the affection will aid in the diagnosis. Again, the palsy may be monocular, and involve all the branches of the third nerve; this is rare, and, if the sixth and fourth be not also paralyzed, the diagnosis may be impossible, unless the further development of the case enable it to be made.

Pflüger publishes[1] a unique case of almost isolated paralysis of each superior oblique, which he diagnosed to be of nuclear origin.

Absolute, or almost absolute, loss of motion of one eyeball may be caused by thrombosis of the cavernous sinus, which may subsequently spread to the opposite cavernous sinus, causing a similar loss of motion in the other eye. The diagnosis between this condition and ophthalmoplegia proper depends on concomitant signs of thrombosis of the sinus (proptosis, œdema, optic neuritis, etc., see page 605) and on the loss of sensation in the ophthalmic division of the fifth nerve, which occurs in cases of thrombosis, but not in those of ophthalmoplegia.

The two following symptoms,—conjugate lateral paralysis and paralysis of convergence,—although not usually included in the term ophthalmoplegia, are in truth often, if not always, caused by nuclear disease.

*Conjugate Lateral Paralysis* (not to be confused with conjugate deviation, page 583).—By this term is meant loss of the power of moving the eyes consensually,—to the right or to the left,—while all other ocular motions, including the power of convergence, are retained. Conjugate lateral pa-

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[1] Archiv für Ophthalmologie, xxxvii., 8, S. 71.

ralysis seems to be the only form of conjugate paralysis which is caused by primary nuclear disease. The vertical forms, other motions of the eye being retained, have been noted, so far as the writer is aware, only with tumors of the quadrigeminal region (page 598).

The prevailing view as to the mode of government of the lateral motions of the eyeballs by the nuclei of the oculo-motor nerves is that, for the purpose of these motions, the nucleus of the sixth nerve on either side governs not only the action of the external rectus of the same side, but also the action of the internal rectus of the opposite eye, and that this is effected by means of communicating fibres between the sixth nucleus of one side and the third nucleus of the opposite side, the existence of which in the posterior longitudinal fasciculus was demonstrated by Graux.[1]

Conjugate lateral paralysis is held, therefore, to be caused by a lesion of the sixth nucleus. Bennett and Saville have recorded[2] a typical case, in which the patient suddenly became affected with loss of power of the associated lateral motion of the eyeballs to the left,—left external rectus and right internal rectus,—with consequent diviation of the eyes to the right, while the power of associated action of the right internus with the left internus for the purpose of convergence, and its power of moving the right eye to the left when the left eye was closed, were perfectly maintained. But the left external rectus was completely paralyzed for all purposes. (In some cases the implicated external rectus maintains its isolated action when the other eye is closed, and the power of convergence is not retained.) The case ended fatally, owing to other complications, and at the post-mortem examination a small patch of softening was found to occupy the position of the left sixth nucleus.

Other than true nuclear, or even pontine, lesions may be the cause of conjugate lateral paralysis as a distant symptom. Thus, for instance, in a case mentioned by Oppenheim,[3] a tumor springing from the basal surface of the left cerebellar hemisphere pressed upon the left side of the pons, and caused conjugate paralysis, the eyes being turned to the right.

In conjugate paralysis, then, the deviation is such that the eyes look away from the lesion, the opposite of what occurs in conjugate deviation due to cortical lesions. In irritative nuclear lesions the eyes must look towards the side of the lesion. Diplopia is rarely present, and then not in a marked degree.

But, if the symptom of lesion of the sixth nucleus be conjugate paralysis, the same lesion cannot cause simple paralysis of the external rectus, as many authors hold it to be capable of doing. Bennett and Saville suggest, that in what is called nuclear paralysis of the sixth nerve, affecting that nerve on its own side alone, the lesion is not in the nucleus, but in the efferent fibres from the nucleus to the external rectus. Knies suggests,

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[1] De la paralysie du moteur oculaire externe, avec déviation conjugée, Paris, 1889.

[2] Brain, July, 1889.

[3] Lehrbuch der Nervenkrankheiten, Berlin, 1894, S. 542.

what is less likely, that nuclear paralysis of the sixth nerve is really due to a lesion at the nucleus, but that conjugate paralysis is due to a lesion in the communicating fibres between the sixth and third nuclei.

In conjugate paralysis, therefore, the internal rectus of one eye, although inactive to either the voluntary or the reflex impulse of conjugate motion emanating from the sixth nucleus of the opposite side, acts in all other respects normally,—because the muscle remains in connection with its own third-nerve nucleus and with the nuclear centre for convergence,—yet sometimes, along with loss of the conjugate lateral motions of the eyes, the power of convergence is lost, and in such cases disease of the nucleus for convergence must be present.

That form of ophthalmoplegia which consists in loss of the lateral motions of the eye to each side is nothing more than double conjugate lateral paralysis due to disease of each sixth nerve nucleus. A case in which this diagnosis was made is recorded by Julius Wolff.[1]

*Paralysis of the Power of Convergence.*—There can be little doubt that a special nucleus in the aqueduct of Sylvius governs the act of convergence of the visual lines. This is probably (Knies) the nucleus which in Perlia's diagram[2] is represented, unlike the other nuclei, as a single and not a double nucleus, lying in the middle line between and close to the nuclei for the ciliary muscles, and not far from those for the sphincters of the pupils. A stimulus from this nucleus would presumably—the fact has yet to be definitely ascertained—throw the two internal recti muscles into simultaneous action. Its corresponding cortical centre would be the macular centre. Lesion of the convergence nucleus would cause loss of the power of convergence, without interfering with the response of the interni to a stimulus coming from their own nuclei, or for the conjugate lateral motions from the sixth nucleus.

In paralysis of convergence, although the eyes together and separately can be moved to the utmost limit of normal lateral motion towards each side, yet if the patient be called on to look at the surgeon's finger held up in the middle line while it is brought gradually closer to his eyes, it will be found that he cannot maintain the fixation, but that, owing to want of power of convergence, the eyes remain in the parallel position. The loss of power of convergence may be complete or partial. There is some homonymous diplopia in every part of the field of fixation.

The accommodation is completely or partially paralyzed in these cases, and it is usually defective even if one eye be closed, showing that the imperfection of the accommodation cannot be regarded as dependent on the paralysis of convergence alone. There is no mydriasis, but the pupillary reflex on attempted convergence is wanting, while the reflex to light is maintained. Difficulty in estimating distances, in consequence of the loss

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[1] Archiv für Augenheilkunde, 1898, xxxvi. S. 257.

[2] Von Graefe's Archiv für Ophthalmologie, xxxv., 4, S. 297.

of power of convergence and accommodation, is usually present. Vertigo was noted by Parinaud[1] as being often complained of by patients with this symptom, and other authors since him have shown that it does not depend on the motor disturbance. It is difficult to offer an explanation of it. That proposed by Parinaud, that the lesion causing the loss of convergence power is in the cerebellum, must now be abandoned.

This paralysis of convergence is quite distinct from insufficiency of the internal recti muscles found in connection with myopia, etc. Loss of converging power is often present in exophthalmic goitre, but must be referred to a mechanical cause rather than to paralysis properly so called.

Eales has recorded[2] a very interesting example of this symptom in a child of thirteen, who presented no other evidence of disease, and in whom recovery took place after a number of years.

Parinaud[3] and Uhthoff[4] point out that paralysis of the power of convergence, partial or complete, without loss of lateral motion, is a form of nuclear palsy which is relatively frequent in disseminated sclerosis.[5]

A case of spasm of convergence is recorded by Berry.[6] It seems probable that it was an hysterical affection.

*Spasm of Ocular Muscles due probably to Pontine Lesions.*—This is the most convenient place at which to refer to this rare symptom. Gowers has recorded[7] two cases of spasm of the external rectus, causing the eye to move towards the external canthus while its fellow remained still, the attack lasting a few seconds. The seat of the disease was probably in the pons, but its exact position and nature are uncertain. Gowers also records[8] a case of Bright's disease in which, with other symptoms indicating a lesion of the pons, there was conjugate paralysis, the eyes being turned to the left. From time to time they were turned still farther to the left, and at the same time agitated by violent nystagmus, in which the quick movement was to the left, the slow return to the right. After death a lesion on the right side of the pons was found. Gowers thinks that the increased paroxysmal movement to the left, and the quick nystagmic movement in the same direction, are to be regarded as liberated from the unaffected left side of the pons, under the influence of the lesion of the right side.

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[1] Brain, ix. p. 330.

[2] Transactions of the Ophthalmological Society of the United Kingdom, iv. p. 300, and xiii. p. 273.

[3] Paralysie de la convergence, Le Progrès médical, 8 Mai, 1886.

[4] Archiv für Psychiatrie und Nervenkrankheiten, xxi. p. 382.

[5] In addition to those mentioned, the following references on this subject may be cited: Parinaud, Bulletins et mémoires de la Société française d'Ophtalmologie, 1886 and 1889, and Archives de neurologie, Mars, 1883; Alfred Graefe, International Ophthalmological Congress, 1888; Stölting und Bruns, Archiv für Ophthalmologie, xxxiv, 3, S. 92, and Neurologisches Centralblatt, 1888, November 15; Peters, Centralblatt für praktische Augenheilkunde, 1889, S. 225.

[6] Transactions of the Ophthalmological Society of the United Kingdom, vii. p. 315.

[7] Ibidem, iv. p. 307.

[8] Loco citato.

*Tumors of the corpora quadrigemina*, or of the quadrigeminal region, especially of the pineal gland, owing to the pressure they are liable to exercise on the central gray matter of the aqueduct of Sylvius, are apt to cause loss of motion of the eyeball very similar to that of primary ophthalmoplegia; and here, then, we have a distant symptom which may render important service in local diagnosis. Nothnagel[1] has pointed out that vertigo, or a reeling gait, is a constant symptom of such disease of the whole quadrigeminal mass,—if the posterior part remain uninjured, disturbances of co-ordination are absent,—and is of opinion that the derangements of co-ordination are the result of the lesion of the quadrigeminal bodies themselves, and are not caused by involvement of the median region of the cerebellum, or by an attendant hydrops ventriculorum. This vertigo is not, of course, pathognomonic of disease of the corpora quadrigemina; it may occur in diseases of the vermiform process, of the pons, of the corpus callosum, in hydrocephalus, in some large tumors of the cerebral hemisphere with great augmentation of intra-cranial pressure, etc. Nothnagel would attach a diagnostic meaning to it only when it appears as the first symptom; for then, as a rule, the point for decision will be, whether the lesion occupies the vermiform process or the corpora quadrigemina. He would decide in favor of the latter position if paralysis of the ocular nerves, especially of the third nerves, subsequently appeared. Yet, that ataxy followed by oculo-motor paralysis in both eyes is not pathognomonic of tumors of the region of the corpora quadrigemina, is shown by a case of Bruns's,[2] in which these symptoms in this sequence were present with a tumor in the cerebellum; while in another case, in which Bruns found a tumor in the quadrigeminal region, the oculo-motor symptoms preceded the static ataxy by a considerable interval. Bruns, consequently, in opposition to Nothnagel, inclines to think, that if the ataxy is the first symptom, the diagnosis is in favor of a cerebellar tumor. He formulates his views, with all reserve, on this difficult diagnostic point as follows: 1. The connection of double ophthalmoplegia with ataxy does not possess the pathognomonic value ascribed to it by Nothnagel as a sign of disease of the corpora quadrigemina, but may be found with a tumor elsewhere,—*e.g*, in the cerebellum. 2. Possibly, if ataxy be present at the commencement and remain a predominant symptom, it may speak for the seat of the lesion being in the cerebellum; while a commencement with ophthalmoplegia, and a predominance of this symptom all through, may indicate quadrigeminal lesion. Yet this is stated with reserve. 3. Should the ophthalmoplegia continue confined to the third and fourth nerves, it seems to speak rather for quadrigeminal disease; but, doubtless, with disease in either locality the sixth nerve may also become implicated. Paralyses of other cranial nerves point to the cerebellum as the *locus morbi*. 4. Intention tremor and choreic motions indicate quadrigeminal disease.

[1] Brain, July, 1889.

[2] Archiv für Psychiatrie und Nervenkrankheiten, 1894, xxvi. S. 224.

The special characters of the ophthalmoplegia in these cases are, Nothnagel states, inequality in the degree of the paralysis, especially in the early period, and inequality in the extent of its distribution. Usually a wide difference between the mobility of the two eyes can be detected, some certain movement of one globe being merely defective, while in the other it is totally annulled. In the later stages the paralysis may be equal bilaterally. Furthermore, it is usual for only some of the third-nerve nuclei to be affected, most commonly those related to the superior and inferior recti; occasionally the lateral movements of the eye are quite abolished, or ptosis may be the first and most marked symptom. It may also happen that the eye is almost completely motionless, as in primary atrophic nuclear paralysis of the ocular nerves; but Nothnagel has never observed in the ophthalmoplegia accompanying tumor of the quadrigeminal bodies such entire immobility of the eyes as sometimes occurs in the primary affection. Death probably takes place before its complete development. Sometimes nystagmus without paralysis of the ocular muscles has been observed. Whether an isolated palsy of the fourth or sixth nerves, with the defect of gait, can claim a diagnostic meaning in the same sense is not yet determined. With cerebral ataxy and ophthalmoplegia, the diagnosis of a lesion of the quadrigeminal bodies would not be shaken even if, some time later, hemiplegia or hemianæsthesia should appear, for these latter symptoms would be explained by pressure on the cerebral peduncle.

Occasionally the ocular muscles in quadrigeminal tumor cases are not paralyzed, nor even paretic.

In a case of tumor of the corpora quadrigemina, Nothnagel[1] observed that remarkable symptom instances of which, although without autopsies, had been already recorded by some other authors (pp. 621 and 622),—namely, a persistent dropping of watery fluid from one nostril.

Conjugate paralysis of the upward movement of the eyes is a rare symptom, and seems to indicate a lesion of the quadrigeminal region. Gowers has recorded a case[2] in which this symptom was present, and in which a small tumor was found in the middle line behind the posterior quadrigeminal bodies, damaging these slightly, the velum, and the adjacent parts of the inferior vermiform process of the cerebellum. He points out that it should be remembered, that disease of the nerves or their roots may chance to affect only the fibres for the superior recti. This was apparently the case in a patient of Thomsen's[3] with an interpeduncular syphiloma. One superior rectus was more affected than the other, a character, Gowers thinks, which is probably of diagnostic importance. In a case of tubercle of the corpora quadrigemina, Henoch[4] observed loss of power of the upward motion of

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[1] Wiener medizinische Blätter, 1888, Nrs. 6–8.

[2] Transactions of the Ophthalmological Society, i. p. 117; Diseases of the Nervous System, 1893, i. p. 185.

[3] Berliner Gesellschaft für Psychiatrie, June, 1886.

[4] Berliner klinische Wochenschrift, 1864, Nr. 13.

the eyeballs as the first focal symptom to appear; and Steffen published[1] a similar case. Paralysis both of the upward and of the downward motion of the eyes, sometimes with ptosis, while the lateral motions are unimpaired, has also been observed, and Gowers thinks it is probably also due to a lesion in the quadrigeminal region. The symptom has been caused, too, by disease in the corpus striatum and optic thalamus.[2] Lang and W. A. Fitzgerald reported a case to the Ophthalmological Society[3] in which this symptom and hemianopsia were the two focal signs. The case recovered, leaving only homonymous insular scotomata. The most recent case on record is Sharkey's.[4]

It is tolerably certain that the loss of power of the upward or of the downward motion of the two eyeballs, or of both of these motions, in these cases of tumor of the quadrigeminal bodies is a distant symptom, yet one of diagnostic value, and does not indicate the presence in these bodies of a centre for those motions analogous to the centre for conjugate lateral motions in the nucleus of the sixth nerve. In Bruns's case of tumor of the corpora quadrigemina[5] the upward and downward motions were intact, while in his case of tumor of the cerebellum there was complete paralysis of the downward motion of each eyeball. In some way which we do not yet understand,—it may be by pressure or otherwise,—tumors of the quadrigeminal bodies sometimes, and also, though more rarely, tumors of the cerebellum, are competent so to act on the nuclei in the aqueduct of Sylvius which govern these motions as to paralyze them without interfering with the powers of neighboring nuclei. The derangements of sight said to be caused by lesions of the quadrigeminal region are treated of on pages 570, 571.

A symptom which is the very opposite of conjugate deviation or paralysis may be best referred to here. It consists in deviation of one eye downward and outward while its fellow is turned upward and inward. This remarkable and as yet wholly inexplicable symptom has been seen only with lesion of the middle cerebellar peduncle, and the lesion may or may not implicate the neighboring cerebellar substance.

3. Fascicular or Radicular Paralysis of the Ocular Nerves.—Fascicular paralysis of the third, fifth, sixth, and seventh nerves is commonly known under its clinical title of crossed or alternate paralysis, a name which it owes to the fact that it is liable to be associated with hemiplegia of the opposite side of the body.

*Fascicular Paralysis of the Third Nerve.*—It may not be possible to make the diagnosis of the lesion causing this symptom if it be confined to the tegmentum of the crus cerebri, from a nuclear lesion. Some or all of the muscles supplied by the third nerve in one eye, or, if the lesion be more extensive, in both eyes, may be deprived of power, but both eyes are rarely

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[1] Berliner klinische Wochenschrift, 1864, Nr. 20.

[2] Wernicke, ibidem, 1876, S. 894, und 1878, S. 154; quoted after Gowers.

[3] Transactions of the Ophthalmological Society of the United Kingdom, ii. p. 230.

[4] Brain, Summer Number, 1894, p. 238.

[5] Archiv für Psychiatrie und Nervenkrankheiten, xxvi. S. 299.

affected symmetrically, as is apt to occur in true nuclear palsy. Allen Starr has pointed out[1] that vertigo is very common in these cases, and he refers the symptom to implication of the red nucleus in the tegmentum—a nucleus which is closely connected with the superior peduncle of the cerebellum—rather than to the diplopia, for the patients complain of vertigo in the dark and when the eyes are closed, as well as in the light and when the eyes are open. Starr thinks, therefore, that it is warrantable to conclude that in partial ophthalmoplegia externa associated with vertigo, which latter is not ocular in origin, the diagnosis of a lesion affecting the red nucleus in the tegmentum cruris may be made.

But if the lesion implicate the pyramidal fibres in the crusta, along with the roots of the third nerve, then the well-known symptom of crossed hemiplegia is produced,—more or less complete loss of power in the third nerve on the side of the lesion, with hemiplegia and paralysis of the lower part of the face of the opposite side of the body. The paralysis of the cranial nerve is usually complete, but it is possible even for one muscle alone to be paralyzed. Leube, for example, has published[2] a case of disease of the peduncle in which the levator palpebræ alone had lost power, ptosis with hemiplegia of the opposite side of the body being the only localizing symptoms; and a similar case has been recorded by Rickards.[3]

Hughlings Jackson has pointed out, that the localizing value of crossed hemiplegia depends chiefly on the hemiplegia and the paralysis of the cranial nerve coming on simultaneously. If they occur at different times, they may be due to two distinct lesions, neither of which may be in the crus; for the hemiplegia may be due to a lesion in the hemisphere, and the third-nerve paralysis to a basal lesion of earlier or later date. But cases have been observed in which, with a lesion in the crus, the third-nerve paralysis preceded the hemiplegia by a considerable interval.

When the lesion causing this crossed paralysis is a tumor, hemianopsia from pressure on the optic tract underlying the peduncle is liable to be an associated symptom.

*Fascicular Paralysis of the Fourth Nerve.*—This is rare. It is almost synonymous with a lesion of the valve of Vieussens, and, consequently, each fourth nerve is apt to be implicated. Solitary paralysis of the fourth nerve due to fascicular lesion is even rarer. The symptom is more likely to be the result of a basal lesion, although then also seldom seen. But Nieden has recorded a case,[4] in which paralysis of one fourth nerve was the only focal symptom to which a tumor of the pineal gland of the size of a walnut gave rise.

Pfungen[5] has pointed out that, in meningitis, exudation in the space be-

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[1] The Journal of Nervous and Mental Disease, May, 1888, p. 315.

[2] Deutsches Archiv für klinische Medicin, xl., 2, S. 317.

[3] British Medical Journal, 1886, i. p. 774.

[4] Neurologisches Centralblatt, 1879, Nr. 8.

[5] Wiener medizinische Blätter, Nrs. 8–11, 1863.

tween the corpora quadrigemina and the splenium of the corpus callosum may implicate the fourth nerves in the valve of Vieussens, and believes it is prone to do so in tubercular meningitis. In combination with paralysis of the third nerve, paralysis of one fourth nerve indicates a lesion in the cerebral peduncle extending back to the valve of Vieussens.

*Fascicular Paralysis of the Sixth Nerve.*—Paralysis of the sixth nerve simultaneous in its onset with hemiplegia of the opposite side of the body indicates a lesion in the pons, usually a hemorrhage, on the side corresponding to the paralyzed nerve. We know that the fifth, the facial, and sometimes the auditory, spinal accessory, and hypoglossal nerves, may all, in varying combinations, form one of the elements in a crossed paralysis from a lesion in this region; but, in the opinion of Nothnagel,[1] if special localizing value is to be here given to the participation of any one cranial nerve, that nerve is the sixth. Paralysis of the facial with the sixth nerve is not an uncommon combination caused by a lesion in the pons, which at the same time produces hemiplegia of the opposite side of the body. This combination is a natural one, in view of the manner in which the fibres of the facial nerve wind round the sixth-nerve nucleus.

*Fascicular Paralysis of the Seventh Nerve.*—Lagophthalmos, we know, is the eye-symptom to which paralysis of the facial nerve gives rise. It is useful for localization, inasmuch as it assists in differentiating a lesion in the internal capsule, or in the facial motor centre of the cortex, from one implicating the portio dura in the pons, as it is absent or very slight in the former cases, but very often markedly present in the latter. This would seem to point to a separate nucleus for the nervous supply of the frontalis and orbicularis palpebrarum muscles. With a lesion in the lower part of the pons, we are apt to have lagophthalmos with crossed hemiplegia; but if the legion be in the upper part of the pons, the fibres from the opposite side having here joined the motor tract, the hemiplegia and lagophthalmos will be homonymous.

*Fascicular Paralysis of the Fifth Nerve.*—As with the sixth nerve, so, too, crossed hemiplegia, in which this paralysis forms a factor, points to disease in the pons. Moreover, when the fibres or nucleus of the nerve in the pons are diseased, neuro-paralytic ophthalmia rarely supervenes, although, no doubt, it sometimes does so; while if the lesion be basal the corneal affection is the rule.

*Absence of Symptoms in Lesions of the Pons.*—It is remarkable that extensive tumors of the pons may be sometimes accompanied by but few ocular paralyses. Thus, in a case of Dr. Finny's, which he kindly asked the writer to see, the external recti were the only ocular muscles paralyzed, although a tubercular tumor occupied the whole thickness of the pons and bulged into the fourth ventricle.[2]

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[1] Loco citato, p. 159.

[2] See also a case reported by Spitzka, The Journal of Nervous and Mental Disease, xiii. p. 193.

It is even possible for a pontine lesion of considerable dimensions, especially if situated in the upper part, to cause no paralysis whatever of cerebral nerves; as in a case of Nothnagel's,[1] where a patch of softening the size of a hazel-nut, situated in the right side of the pons, in the centre of its thickness, and in its upper half, had left hemiplegia as its only focal symptom. Such cases cannot be distinguished from those caused by lesion in the internal capsule.

Paralyses confined to cerebral nerves, in cases of pure pontine lesion, are rare; yet such cases do occur,[2] and, consequently, for the diagnosis of disease of the pons, paralysis of the extremities is not absolutely necessary.

Finally, lesions of the pons, especially tubercular tumors, may be latent, not giving rise to paralysis either of cerebral nerves or of the extremities. Many cases are on record[3] in which large tumors were found in the pons, after death, which had resulted in consequence of disease elsewhere. In a case of Ladame's, quoted by Nothnagel, a sarcomatous tumor occupied nearly the entire pons without giving rise to any marked motor or sensitive paralysis. The only explanation which Ladame or Nothnagel can suggest for this extraordinary circumstance is, that the slowly growing tumor did not rupture nor destroy the nerve-fibres, but merely pressed them together. Yet the conducting function of nerve-fibres is usually easily interfered with by pressure.

*Pupillary Symptoms in Lesions of the Pons.*—Bilateral myosis is often seen with hemorrhage in the pons, and is usually reckoned among the more important symptoms of the lesion; but it is by no means a constant symptom of it, the pupils here being frequently of normal size. Moreover, although myosis is most commonly the result of pontine lesions, yet it may accompany disease in other parts, as, for instance, hemorrhage in the corpus striatum, which bursts into the lateral ventricle, and in meningeal hemorrhages. Indeed, the condition of the pupils, whether normal, contracted, or dilated, is of very slight significance or value in the diagnosis either of the position or of the kind of any intra-cranial lesion. Bilateral mydriasis is frequently present in apoplectic coma, without reference to any particular locality, and the same holds good as regards monolateral myosis and mydriasis. And yet, slight in value as is the state of the pupils in cerebral cases, there is probably no symptom which receives more regular attention from the clinicist in his note-taking. Of course, where the third nerve is believed to be injured, either at its nucleus or in its course, pupillary symptoms have a distinct value.

4. Paralysis of the Orbital Muscles, of the Internal Muscles of the Eye, and of the Fifth Nerve, due to Lesions at the Base of the Brain.—(The following pages should be read in connection with those which treat of lesions of the optic tract and optic commissure,

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[1] Topische Diagnostik der Gehirnkrankheiten, S. 107.
[2] Nothnagel, loco citato, p. 125.
[3] Ibidem, p. 114.

pp. 572 and 576.) An attempt to give a systematic account of the eye-symptoms caused by lesions at the base of the brain, which will be true to nature as we find it in the cases that come before us, is even a more difficult task than that presented by eye-symptoms caused by other cerebral lesions, and can be successful in a limited sense only. Basal lesions may, according to their extent and situation, involve one, two, or more of the many cranial nerve-trunks in this region, as well as the optic tracts, the optic commissure, and the cerebral peduncles. Even the natural rule, which renders structures lying close together apt to be attacked by one and the same lesion, is subject to many exceptions in basal lesions, especially in syphilitic disease, mainly gummatous exudation, where we not rarely see an extensive lesion producing symptoms derived from widely separated structures, without interference with the functions of those which intervene. Indeed, one of the chief diagnostic features in basal lesions is their great tendency to implicate several different nerve-trunks, without reference to system or function.

It must, however, be remembered that other than basal disease, in the strict sense of the term, may cause basal eye-symptoms; for tumors of the hemispheres, or cerebellum, may grow towards the base until they finally implicate directly the nerve-trunks and other structures there. This is especially liable to occur with tumors of the frontal lobe which tend much to grow towards the base. Hence among the later symptoms of frontal-lobe tumors one not rarely sees blindness of one eye, or it may be hemianopsia and paralysis of the sixth or third nerves, associated with monolateral anosmia. The history of the progress of the symptoms can alone in such a case render a correct diagnosis possible. Moreover, paralysis of cerebral nerves at the base is not uncommon as a distant symptom in tumors of the hemispheres and cerebellum,—a fact which should be borne in mind in the localizing of these lesions. The sixth nerve seems to be that one most likely to be affected in this manner, and this is probably due to the circumstance that it is much exposed to the effects of pressure where it curves round the margin of the dorsum sellæ.

It will be convenient to consider the basal paralyses of the motor eye-nerves and of the first division of the fifth nerve *seriatim.*

*Third-Nerve Basal Paralysis.*—This nerve is most likely to become implicated by disease either in the interpeduncular space or at the cavernous sinus.

Double third-nerve paralysis is not infrequent in basal lesions, and in those instances the interpeduncular space may be said to be invariably the starting-point of the disease.

Paralysis of one or both sides of the body, from implication of the peduncles in the disease, is not a rare accompaniment; or, if one optic tract be implicated, homonymous hemianopsia will be present; if both tracts, then total blindness of each eye is found. But it should be remembered that the hemianopsia may be due to a separate lesion in one hemisphere, while the third-nerve paralysis is due to basal disease. The hemianopic

pupil may be expected to help in this diagnosis. Double third-nerve paralysis rarely occurs without paralysis of the fourth, fifth, sixth, or seventh, —one or all; yet complete double third-nerve paralysis without any complication may result from a basal lesion, and may cause a difficulty of diagnosis as to whether there is a basal or a nuclear origin for the motor disturbances which cannot at once be cleared up. Still more likely to give rise to an error in diagnosis of this kind are those rare cases of partial paralysis of each third nerve due to basal disease, in which the symmetrical distribution of the loss of motion is very suggestive of nuclear paralysis, especially if uncomplicated by other basal affections. The following, for example, have been observed as the result of basal disease of each third nerve: slight double ptosis, with dilatation of one pupil; double ptosis and paralysis of each internal rectus; loss of the upward and downward motions of each eye, with retention of the lateral motions,[1] in a case where a gumma in the interpeduncular space was the diseased condition; paralysis of each internal rectus, caused by a small interpeduncular hemorrhage in a case of hemorrhagic pachymeningitis of the convexity of the left hemisphere;[2] loss of the upward motion only of each eye, with a gumma in the same position.[3] Isolated ophthalmoplegia interna, double or single, has not been observed in basal third-nerve lesions.

Monocular third-nerve paralysis from basal disease may also be complete or partial, uncomplicated or complicated. Complete third-nerve basal paralysis of one eye is more common than its partial paralysis. In the partial cases it is possible that the levator palpebræ may be the only muscle paralyzed; and that isolated monocular ptosis may be caused by third-nerve lesion at the base of the brain is an important fact, in view of the opinion which has been held, that this symptom occurs with cortical lesions only.

Paralysis of one third nerve, complete or partial, with hemiplegia of the opposite side of the body,—the most usual form of crossed paralysis,—may be caused by a basal lesion involving the trunk of the third nerve and the corresponding peduncle near the pons; but it must be borne in mind, that a lesion in the motor path in the hemisphere may be the cause of the hemiplegia, while the third-nerve paralysis may be due to the basal lesion. Yet the latter arrangement is more rare than a single basal lesion as the common cause of both symptoms.

Crossed hemiplegia, whether it be the third or some other nerve which is concerned, due to lesions at the base, cannot always be distinguished, when uncomplicated by obvious basal symptoms, from a crossed hemiplegia due to pontine lesions. The main point in the diagnosis would be the mode of onset of the symptoms. If both symptoms come on suddenly and simultaneously, the assumption is that the lesion is pontine; but if gradually and in succession, the lesion is more apt to be basal.

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[1] Uhthoff, Archiv für Ophthalmologie, xl. 1.

[2] Bouchaud, Revue de médecine, 1891, p. 595.

[3] Thomsen, Centralblatt für praktische Augenheilkunde, 1886, S. 203.

Paralysis of one third nerve may also be complicated with disease of the corresponding optic tract and resulting hemianopsia. Tumors and inflammatory products about the cavernous sinus are very liable to cause partial or complete third-nerve paralysis, along with partial or complete paralysis of some or all of the other orbital nerves, as well as of the optic and olfactory nerves.

Paralysis of the third nerve in cases of fracture of the base of the skull is by no means uncommon. If the line of fracture be such as to lacerate the nerve near its apparent origin close to the anterior margin of the pons, both nerves are apt to be injured owing to their proximity, and there may be no damage to the optic tract or nerve. When the fracture passes through the sphenoid bone, in the neighborhood of the sphenoidal fissure and optic foramen, only one third nerve is liable to injury, and blindness of the corresponding eye from trauma of the optic nerve is frequently present.

*Thrombosis of the Cavernous Sinus.*—Thrombosis of the cavernous sinus is one of the most important of the diseased conditions which affect this part, and paralysis of the third nerve is one of its most constant symptoms. It is mainly inflammatory thrombosis which is here in question, the marasmic form seldom affecting the cavernous sinus.[1] Its principal causes are: infective inflammation of the orbital cavity, passing backward through the ophthalmic veins, or along the sheaths of the nerves, and inducing meningitis; erysipelas of the face, followed by phlebitis of the angular and ophthalmic veins; infective inflammation in the buccal, nasal, and pharyngeal cavities, and of the body of the sphenoid; and extension of thrombosis from the lateral, longitudinal, or petrosal sinus, especially in cases of purulent otitis. The cavernous sinus may be said never to undergo primary thrombosis. In considering the symptoms of thrombosis of the cavernous sinus, Macewen says it ought to be borne in mind, that although at the outset only one cavernous sinus is affected, yet the thrombosis in more than half the cases spreads through the circular sinus to the cavernous sinus on the opposite side. Then the side on which the symptoms began may be partially restored, while the other side becomes markedly affected; and this alternation, when present, is a diagnostic sign between orbital inflammations and cavernous sinus thrombosis. When the invasion occurs from the intra-cranial direction, pain in one or all of the branches of the first division of the fifth pair is usually an early symptom. There is also exophthalmos depending on venous obstruction, which may subside considerably when collateral circulation is established. Should there be ophthalmic phlebitis and retro-orbital abscess, the proptosis is more marked. Œdema of the lids, and chemosis from the venous stasis, are also often present. Congestion papilla is sometimes found, but by no means constantly so, thanks to the free anastomosis of the superior ophthalmic vein

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[1] Knapp, Archiv für Ophthalmologie, xiv., 1, S. 220; Macewen, Pyogenic Diseases of the Brain and Spinal Cord, Glasgow, 1893, p. 245.

with the angular and supra-orbital veins. Probably its occurrence depends mainly on the presence of a thrombus in the ophthalmic vein. All the orbital nerves, as well as the fifth nerve and the optic nerve, are liable to be involved, giving rise to complete, or almost complete loss of all the motions of the eyeball, with loss of sensation in the conjunctiva and cornea, and blindness of the affected eye. The pupil is usually contracted at the beginning, but at a later period becomes dilated. Along with these local symptoms there are the general ones of rigors, high temperature, and vomiting. The disease may last from a few days to several months, but always ends fatally in the cases of inflammatory thrombosis.

*New Growth occupying the Cavernous Sinus.*—Nettleship[1] publishes a case in which a sarcomatous tumor occupied the right cavernous sinus and sella turcica. The third nerve passed along the upper border of the growth, was infiltrated by it, and had become atrophied by pressure. The sixth nerve pierced the tumor and was also atrophied. During life there was total ophthalmoplegia externa and interna, complete ptosis, and slight proptosis. There were no ophthalmoscopic changes. The optic and fifth nerves were not affected, and vision was good.

*Aneurism of the Ophthalmic Artery near its Origin, Aneurism of the Internal Carotid in the Cavernous Sinus, and Rupture of the Internal Carotid in the Cavernous Sinus.*—These are vascular diseases of this region which cause loss of power in the motions of the eyeballs, as well as other striking eye-symptoms, and it is convenient to draw attention to them here; but a detailed account of these conditions does not come within the scope of this chapter, and therefore their symptoms only will be briefly noted. The symptoms of aneurism of the ophthalmic artery near its origin, and of the internal carotid in the cavernous sinus, are not very different, and in most cases it is not possible to make the diagnosis. They both are rare, aneurism of the internal carotid being the more common of the two. In aneurism of the ophthalmic artery there is blindness of the corresponding eye, due to interference with the blood-supply of the retina and to direct pressure on the optic nerve by the aneurism. There is also exophthalmos from œdema of the orbital tissues and distention of the ophthalmic vein, with chemosis and swelling of the eyelids,—all the result of venous obstruction from pressure of the aneurism on the ophthalmic vein and cavernous sinus. The exophthalmos may be pulsating, by transmission of pulsations of the aneurism to the column of blood in the distended vein and sinus; but the pulsation may be absent, and when present is usually slight. Loss of power in the muscles supplied by the third and sixth nerves, from pressure on these nerves by the swollen ophthalmic vein, is also often present. Noises in the head are complained of by the patient, and can be objectively discerned by auscultation. They cease on compression of the common carotid.

In aneurism of the internal carotid in the cavernous sinus the loss

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[1] Transactions of the Ophthalmological Society of the United Kingdom, i. p. 186.

of the motions of the eyeball is apt to be more marked, and the loss of vision less marked, than in aneurism of the ophthalmic artery; and in the former the ophthalmic division of the fifth nerve is more likely to be implicated. In either case, the more rapidly the aneurism develops the more pronounced the symptoms tend to be, for in a slow and gradual evolution of it the vein, sinus, and nerves become accustomed to its pressure.

Rupture of the internal carotid in the cavernous sinus, either as the result of injury (penetrating wounds, blows on the head, fractures of the base), or of disease of the coats of the vessel, is a condition which, occurring, as it does, suddenly, produces very marked symptoms of venous obstruction and of pressure on surrounding tissues,—the eyelids swollen and livid, the surface of the eyeball covered with distended veins, the conjunctiva chemotic, the eyeball protruded. Pulsation of the protruded eyeball comes on somewhat later (pulsating exophthalmos), but never fails to appear, and is a striking symptom. Distended and varicose veins may, after a time, be felt by the tip of the finger in the orbit and may be seen on the forehead, and even at an early period the ophthalmoscope shows distention of the retinal veins, with occasionally pulsation of them. A continuous subjective and objective bruit, which becomes intensified at each systole, is present. The third and sixth nerves are those most commonly paralyzed, and the ophthalmic division of the fifth nerve is also liable to be affected.

Vision is often not seriously affected at first. After a time, loss of sight may be caused by pressure of the distended vein on the optic nerve, by deficient supply of arterial blood to the retina, by the development of congestion papilla, and by retinal ecchymoses.

*Recurrent Third-Nerve Paralysis.*—Recurrent third-nerve paralysis, without paralysis of any other cerebral nerves, except that in a few instances more or less anæsthesia of the parts supplied by the fifth nerve has been present, is a remarkable symptom, held by many to be due to basal lesions. Only one, and in each case always the same, third nerve is affected. The disease usually begins in childhood or youth, but there are exceptions to this, and it is more common in females than in males. As a rule, all the muscles supplied by the third nerve, inclusive of the internal ocular muscles, lose their power. The intervals between the paralytic attacks vary from ten days to one year, each attack lasting from two or three days to several months. The shorter the attacks of paralysis the more frequently do they recur, and sometimes the paralysis does not completely disappear during the intervals. In most cases severe hemicrania precedes the paralytic attacks, and this is situated always on the side of the paralysis. Nausea, vomiting, rigors, and general malaise often accompany the headache; so that Charcot and others[1] have regarded the affection as migraine complicated by paralysis of the third nerve (migraine ophthalmique or ophthalmoplégique). The headache, nausea, etc., cease as soon as

[1] Le Progrès médical, 1890, Nos. 81, 82.

the paralysis—often suddenly—comes on. Most of the cases have a progressive character, the recurrences of the paralysis becoming more frequent.

The pathogenesis of the affection is obscure. Some authors regard it, when truly periodic, as functional, while the exacerbating cases they take to be due to organic basal disease; but others[1] believe that both forms are caused by organic disease, inflammatory exudation around the nerve-trunk,[2] degeneration of the nerve-trunk,[3] and fibroma[4] and fibro-chondroma of the nerve-trunk[5] having been the changes found in the only four cases out of twenty-nine published in which a post-mortem was made. But while this explains the paralysis, it does not explain its intermissions; and, to account for these, recourse has been had to the not very satisfactory assumption, that alterations in the circulation of the diseased part alternately augment and diminish its pressure on the nerve. In one case,[6] which was clearly due to malarial poisoning, the author assumed an inflammatory process of the trunk of the third nerve as the cause.

Treatment has little, if any, influence in this disease. Charcot used bromide and iodide of potassium, and mercurial inunctions. In Kljatschin's case, due to malarial poisoning, quinine in large doses acted in a markedly beneficial manner.

Recurrent third-nerve paralysis has been seen as a very early initial symptom of approaching general paralysis, and it may occur as an intercurrent symptom of the pronounced state; but it does not foretell impending focal cerebral disease of a serious nature, only one case (Gubler's) having presented serious cerebral symptoms ending in death. It is sometimes a manifestation of hysteria. Holmes Spicer and Armerod[7] record several cases of recurrent attacks of paralysis of the sixth nerve.

*Fourth-Nerve Basal Paralysis.*—This nerve is more rarely affected by basal lesions than the third or the sixth, and still more uncommon are cases in which it is the only nerve implicated. The third nerve is almost always implicated with it, while sixth-nerve paralysis, and optic-tract symptoms, and crossed hemiplegia may form elements in the symptom-complex of the case. Leber[8] reports two cases of falls on the head in which paralysis of one fourth nerve was the main symptom. Probably there was no fracture in either case, and the paralysis was held to be due to rupture of the trunk of the nerve owing to the concussion of the skull.

*Sixth-Nerve Basal Paralysis.*—Monolateral basal paralysis of this nerve is less common than that of the third nerve, yet double sixth-nerve paralysis

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[1] Darkschewitsch, Deutsches Archiv für klinische Medicin, xlix. 4, 5.
[2] Gubler, Gazette des hôpitaux, 1860, No. 17.
[3] Weiss, Wiener medizinische Wochenschrift, April, 1889.
[4] Karplus, ibidem, 1895, Nrs. 50–52.
[5] Richter, Archiv für Psychiatrie und Nervenkrankheiten, 1887.
[6] Kljatschin, Neurologisches Centralblatt, 1897, S. 206.
[7] Transactions of the Ophthalmological Society of the United Kingdom, 1896, p. 286.
[8] Archiv für Ophthalmologie, xxvii., 1, S. 297.

is relatively frequent in basal disease, when the latter is situated centrally in the posterior fossa (when, too, the anterior pyramids of the medulla may be pressed on, with consequent motor symptoms in the limbs), especially in syphilitic affections and in fractures of the base.[1] Monolateral sixth-nerve paralysis is also not infrequently present as a result of fractures of the base.

It is probable that in these fractures the sixth nerve is specially liable to be injured, owing to its intimate connection with the margin of the dorsum sellæ and the periosteum in the neighborhood of the apex of the petrous portion of the temporal bone; which latter, it is stated, is very apt to be cracked in these accidents. It is recognized that sixth-nerve paralysis may be caused by fracture of the base without coma, paralysis of other cranial nerves, or the usual symptoms of basal fractures being present. In such cases it may not be possible to say, especially if both sixth nerves are implicated, whether the lesion is basal or nuclear. If the loss of power is only partial, it points to a nuclear lesion, especially if polyuria is present.[2]

Paralysis of one sixth nerve, with hemiplegia of the opposite side of the body due to implication of the peduncle,—crossed paralysis,—is sometimes seen with the lesion near the posterior margin of the pons, and it is apt to be accompanied by paralysis of the seventh nerve, usually on its own side. Eulenberg[3] records a unique case of isolated traumatic basal paralysis of the sixth nerve, no other cranial nerve having been implicated. The injury was a stab received in the temporal region, which must have divided the trunk of the nerve at the base of the skull, below the posterior clinoid process, without injuring the sinus or the carotid.

*Seventh-Nerve Basal Paralysis producing Lagophthalmos.*—There is practically nothing to be added in this connection to what has been incidentally stated above when treating of basal paralysis of the third, fourth, and sixth nerves. Isolated seventh-nerve paralysis rarely occurs from basal disease. In fractures of the base of the skull the line of fracture frequently passes through the petrous bone, and then the facial nerve may be injured; or it may become paralyzed some days after the accident from the inflammatory process which is set up. In facial paralysis due to cortical lesion, or to any lesion above the nucleus, the orbicularis palpebrarum and other muscles in the upper part of the face are but little, if at all, affected, while it is mainly the muscles which go to the angle of the mouth that are paralyzed; also, emotional movements (in laughing or crying) are often less impaired than voluntary movements. On the other hand, in nuclear, fascicular, and basal facial palsy the orbicularis palpebrarum and the frontal muscle, as well as the muscles of the mouth, become paralyzed, and the emotional and voluntary facial motions suffer equally.

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[1] Purtscher, Archiv für Augenheilkunde, xviii. S. 387.

[2] Purtscher, Archives of Ophthalmology, xxiii., 4, p. 398.

[3] Neurologisches Centralblatt, 1894, S. 578.

Silex[1] has seen three cases of isolated paralysis of the orbicularis of the upper lid, and they are the only cases of the kind on record. They all underwent cure. The cases offer a further proof of the correctness of the view put forward by Mendel,[2] and supported by Tooth and Turner,[3] that the oculofacial group of muscles (frontalis, orbicularis palpebrarum, and corrugator supercilii) are innervated by the third-nerve nucleus, the fibres passing into the trunk of the seventh nerve by way of the posterior longitudinal bundle.

*Fifth-Nerve Basal Paralysis.*—Basal lesions not infrequently cause paralysis of this nerve, combined in most instances with paralysis of the third nerve, and very often with paralysis of the sixth nerve, or with disease of the optic tract; and complication with facial paralysis is also not uncommon. Neuroparalytic keratitis is sometimes, but by no means always, seen in these cases, and the coexistence of ptosis does not necessarily obviate the occurrence of the corneal affection, although it seems to render it less likely to occur. A lesion of the ophthalmic division only of the nerve is present, when the diseased process is at the cavernous sinus or the sphenoidal fissure.

Complication with paralysis of the opposite side of the body sometimes occurs with basal paralysis of the fifth nerve; and sometimes, especially with syphilitic gummata, the hemiplegia is of the same side of the body, indicating implication of the opposite peduncle.

Syphilitic gummatous exudation is the commonest basal lesion causing paralysis of the cranial nerves, and the diagnosis of its presence is supported if there be instability in those paralyses: the defect in the field of vision may be different in degree from day to day, immobility of the pupil may come and go, ptosis and paralysis of the facial may disappear and return again, and so on. This variableness of the symptoms is the result of the rapid growth, followed by rapid shrinkage of the granulation tissue which surrounds the trunks of the nerves.

Basal paralyses are also, we believe, often due to a chronic and indolent form of meningitis of a rheumatic origin; and this, too, when other signs of rheumatism are wanting.

## DIFFUSE DISEASES OF THE BRAIN.

Under this heading it is proposed to give an account of those eye-symptoms which are apt to be present, with more or less constancy, in certain organic diseases of the brain that are known to be diffuse in the sense that they occupy a wide extent of cerebral surface or substance; or which belong to the category of brain-diseases that are believed to be organic, although as yet the nature of the changes, and whether they are local or diffuse, have not been definitely ascertained; or which are in the

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[1] Archiv für Augenheilkunde, 1896, xxxii. S. 95.
[2] Neurologisches Centralblatt, 1887, S. 537.
[3] Brain, Winter Number, 1891, p. 491.

uncertain and ever-diminishing neutral territory between diffuse organic and functional brain-disease. The diseases here to be considered are: disseminated sclerosis, diffuse sclerosis, pseudo-bulbar paralysis of cerebral origin, general paralysis, meningitis, acute hemorrhagic encephalitis, hydrocephalus, infantile paralysis, degeneration of cerebral arteries, amaurotic family idiocy, paralytic vertigo, paralysis agitans, encephalopathia saturnina, epilepsy, and chorea. Eye-symptoms occurring with certain deformities of the cranium will also be mentioned.

Information concerning eye-symptoms due to fractures of the skull has been given above at pp. 581, 605, and 609.

## DISSEMINATED SCLEROSIS OF THE BRAIN AND SPINAL CORD.

The eye-symptoms in this disease are of considerable diagnostic value, and have been subjected to careful and exhaustive studies by Uhthoff,[1] Buzzard,[2] and Parinaud and Marie.[3] They consist in certain visual defects and ophthalmoscopic appearances, and in certain oculo-motor disturbances.

The most common visual defect is amblyopia with central color scotoma. In a small proportion of cases absolute central scotoma is found. Irregular peripheral defects in the field—sometimes only for color—occur, with and without central scotoma, and in other cases a regular concentric contraction of the field takes place. One or both eyes may be affected in any of these ways. Any of these visual disturbances may get better, or may after a time entirely disappear. The onset of the defect of sight is usually very rapid, or almost sudden, but it sometimes comes on gradually. Complete and permanent amaurosis in disseminated sclerosis is very rare indeed; but temporary amaurosis occurs, as Charcot pointed out, and may last as long as five or six months, with, then, a remission of several months, and in some cases many such relapses take place.

The ophthalmoscopic appearances in connection with these visual defects may be absolutely normal; or, on the other hand, any of the ophthalmoscopic changes which will be mentioned may be present with normal vision; or, again, there may be both defect of vision and ophthalmoscopic signs. Hence it is always necessary to examine both the vision (and especially the field of vision) and the fundus oculi. Diseased ophthalmoscopic appearances occur in about one-half of the cases of disseminated sclerosis. Most frequently these changes consist in a simple, and not very intense, atrophic discoloration of the whole surface of the optic papilla; or very often the temporal part of the disk is atrophic, as it is in toxic amblyopia, while its inner two-thirds are normal; yet in these latter cases the observer must not expect always to find a central scotoma, nor, under the

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[1] Archiv für Psychiatrie und Nervenkrankheiten, xxi., 55, S. 303.

[2] Brain, 1890; British Medical Journal, October 7, 1893; Transactions of the Ophthalmological Society of the United Kingdom, 1897, p. 124.

[3] Leçons sur les maladies de la moelle, Paris, 1892.

microscope, are the papillo-macular fasciculi in the trunk of the optic nerve found to be diseased. Only very rarely is there complete atrophic discoloration of the entire optic papilla, so that the inner part of the papilla displays no pink hue whatever. In about five per cent. of the cases optic neuritis is found.[1] Uhthoff thinks that a slight transient neuritis may more frequently occur than is generally supposed, and may leave after it either a normal optic papilla or the commencement of an atrophic discoloration; and he thinks this papillitis is caused by the presence of sclerotizing foci immediately behind the lamina cribrosa. Buzzard, too, thinks neuritis may be present. The ophthalmoscopic changes may be in both eyes or in one only.

Uhthoff found that in disseminated sclerosis there can be marked disease in the trunk of the optic nerve without any abnormal ophthalmoscopic appearance or defect of sight. It would seem that the conducting function of the nerve-fibres need not be interfered with, although in passing through the sclerosed patches they are deprived of their medullary sheaths and reduced to axis-cylinders.

There is, therefore, a remarkable absence of direct relation between the state of vision, the ophthalmoscopic appearances, and the pathological condition of the trunk of the optic nerve, respectively, in this disease.

Hemianopsia has never been observed in disseminated sclerosis; consequently, it is certain that the derangements of vision are due to disease in the optic nerve, and not in the chiasma or the optic tract.[2]

Defects of vision and abnormal ophthalmoscopic appearances, one or both, occasionally precede other symptoms of the disease by some months or even years; in some cases they appear in its early stage, but it is more usual for them to come on after the general symptoms are well developed.

The oculo-motor derangements which may be found in disseminated sclerosis are: isolated paralyses of orbital muscles, nuclear paralyses, and nystagmus. (See also page 590.) Probably some of the isolated paralyses —such as double paralysis of the sixth nerve, and paralysis of some of the branches of the third nerve—are of nuclear origin. Paralysis of single muscles is nearly always partial, and generally disappears in the course of a few weeks. The paralyses concerning the nuclear origin of which there can be no doubt are: loss or defect of the conjugate motions to the right or to the left, and paresis of the power of convergence. Marked exterior ophthalmoplegia has been observed, but is very rare. Kunn[3] has observed the development of true convergent strabismus in some cases.

Nystagmus is present in about one-half of the cases of disseminated sclerosis. It is found either of the ordinary type or as nystagmic twitch-

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[1] Uhthoff, loco citato; Nettleship and Sharkey, Transactions of the Ophthalmological Society of the United Kingdom, iii. p. 225.

[2] Wilfred Harris, however, states that he has seen hemianopsia in disseminated sclerosis. Brain, Autumn Number, 1897, p. 315.

[3] Beiträge zur Augenheilkunde, 1898, Heft xxiii. S. 65.

ings, especially when the eyes are brought into the extreme positions (ataxic nystagmus). Paralyses of muscles may be present along with the nystagmus. True nystagmus is very rare as a symptom in other forms of general nervous disease, therefore it is of much diagnostic value here. Nystagmic twitchings do occur in other diseases of the nervous system, but are infinitely more common in disseminated sclerosis than in any other disease. Very slight twitchings in the extreme positions should not be put down as a diseased symptom, for they may be seen in some healthy eyes. Kunn describes a symptom which he terms fixation trembling, and which takes place, where there is no nystagmus, at the moment the eyes are brought from a position of rest to look sharply at a near object. At the first attempt the visual axes are brought to converge at a point nearer than the desired distance, the next moment the convergence is too slight and the visual axes are directed to a point beyond the object, and finally, after two or three such rapid oscillations, the fixation becomes steady at the correct distance.

The various oculo-motor affections, similarly as the affections of sight and the ophthalmoscopic changes, occur, as a rule, in the course of the pronounced disease, occasionally in its initial stage, and very rarely before any other symptom.

The state of the pupils in disseminated sclerosis is usually normal. Parinaud[1] thought that the activity of the pupil-reflexes to light and accommodation was markedly increased in many cases. In advanced stages there is usually myosis. The Argyll-Robertson pupil occurs very rarely, and inequality of the pupils is occasionally to be noted. The phenomenon of hippus is relatively frequent, and in two cases observed by Damsch[2] it was the only demonstrable cerebral symptom. Kunn noticed in some of his cases, in which there was normal visual acuity, a difficulty in reading (fuzziness and dancing of the letters and lines), which he believes to be due to a trembling of the ciliary muscle.

Hysteria is the disease for which some cases of disseminated sclerosis in their incipient stage are liable to be, and no doubt often are, mistaken, owing to the presence of such symptoms as transitory loss of power in one or more limbs, aphonia, convulsive seizures, hysterical manner, etc., while the symptoms characteristic of disseminated sclerosis are as yet wanting. In such cases the eye-symptoms are capable of giving much aid in the diagnosis. In hysteria the ophthalmoscopic appearances are normal; in disseminated sclerosis in fifty per cent. of the cases there is some change in the optic papilla. In hysteria when the fields of vision are deranged they become contracted and transitory central scotoma is extremely rare; in disseminated sclerosis the fields may become contracted, but central scotoma (often only for colors), which may come and go, is the more common form

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[1] Le Progrès médical, August, 1884.

[2] Neurologisches Centralblatt, 1890, S. 258.

of defect. In hysteria when the fields become contracted the boundaries for color often do not recede in their regular order; for example, the field for red may be wider than that for the other colors, or the color-field boundaries may cross each other irregularly, or else, according to Buzzard and Head, the zone between the outer limit of the field for white and the outer limit of the field for color is obliterated, and white, green, red, and other colors are visible within the same limits; in disseminated sclerosis the contraction of the boundaries for the perception of the several colors in the field, when there is any such contraction, follows that for white in the regular physiological order of the fields, and the zone between the outermost boundary for white- and for color-perception is maintained. In hysteria, too, it may be found impossible to examine the color-fields at all, all colors being styled "dark" or "black" either immediately or in a few moments after the examination of the fields had commenced. In hysteria oculo-motor disturbances very seldom occur, while in disseminated sclerosis nystagmus is common, and paralysis—often transitory—of an orbital muscle not rare.

## DIFFUSE SCLEROSIS OF THE BRAIN.

Both Gowers[1] and B. Bramwell[2] state that occasionally symptoms very similar to those of intra-cranial tumor are seen in this diseased state. The latter author says, "In some very rare and quite exceptional cases, in which the characteristic symptoms of an intra-cranial tumor (headache, vomiting, and double optic neuritis) were present during life, no coarse lesion of the brain, but only sclerosis, atrophy, or microscopical lesions have been found at the post-mortem. In such cases a correct diagnosis is obviously impossible. These cases show that the general symptoms of intra-cranial tumor are not in all cases sufficient for a correct diagnosis, unless localizing symptoms are at the same time present. The most experienced observers will occasionally, in the present state of our knowledge, make mistakes, and fail to find on post-mortem examination the tumor which had been confidently expected during life. It remains to be seen whether a more extended observation and experience will enable a distinction to be made in cases of this description."

## PSEUDO-BULBAR PARALYSIS OF CEREBRAL ORIGIN.

Eye-symptoms in this condition (there is also pseudo-bulbar palsy caused by the pressure of tumors, and so on, of which the diagnosis can only be made by aid of concomitant symptoms of the primary disease) are not very common; yet, when present, they are of service in the diagnosis between this condition and true bulbar paralysis, in which no eye symptoms occur. Optic neuritis or optic atrophy may be found. The voluntary power of closing the eyelids is sometimes wanting, while their reflex closure

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[1] Diseases of the Brain.

[2] Intra-Cranial Tumors, p. 131.

can be effected, but is maintained only for a moment, the eye being opened again directly. Derangements also of the voluntary movements of the eye-balls have been occasionally observed. The eyes in some cases could not be moved to either side by an effort of the will, although they could turn to look at an object held up, or in the direction of an unexpected noise. Again, in some cases, an effort of the will failed at first to turn the eyes in a desired direction, but after an interval of time the voluntary motion was effected.

## GENERAL PARALYSIS OF THE INSANE.

The eye-symptoms which may be present in this disease are: 1. Derangements of the intra-ocular muscles, especially pupillary symptoms. 2. Orbital muscle paralyses. 3. Atrophy of the optic disk. 4. Mind-blindness.

*Pupillary Symptoms, etc.*—The pupillary anomalies are numerous, and, in view of their frequent appearance in the prodromal stage, very important. They consist in myosis, mydriasis, inequality of the pupils, irregular shape, loss of reflex to sensitive stimuli, loss of the direct light-reflex, loss of consensual reflex, and Argyll-Robertson pupil. There is also, sometimes, paralysis of accommodation.

As regards the size of the pupils, they are usually contracted in the early stages and more or less dilated in the later stages. An early sign is a slight, perhaps hardly appreciable, inequality in the size of the pupils, although neither of them may be of obviously abnormal size, the wider of the two reacting rather more sluggishly to light than its fellow. Dawson and Rambaut[1] found this sign in ninety-two per cent. of their cases, yet, as it is not uncommon in other forms of insanity not resembling general paralysis, they do not regard it as an important symptom, and believe that any value it possesses is purely negative. There is apt to be associated with this, or it may be found even earlier, loss of the pupillary reflex to sensitive stimuli, or loss of the "sympathetic dilatation," as it is sometimes called. In later stages of the disease the larger of the two pupils reacts not at all, or but very slightly, to light, although the vision of the eye remains perfect and the pupil of the other eye continues normal. Bevan Lewis[2] has constantly observed that when unilateral paralysis or convulsions occur in the early stages of general paralysis, the dilated pupil is on the side of the paralyzing or discharging lesion. An early indication of commencing iridoplegia is given by focal illumination; for although the pupil is active to the concentrated beam of light, it shows a very limited range of movement, together with an oscillation, or hippus, which then tends to wide dilatation, even under this bright illumination of the retina. This tendency to dilate during stimulation by light (known as the paradoxical pupil-symptom) appears to Lewis to be the earliest augury of coming paralysis.

[1] British Medical Journal, September 10, 1898.

[2] Text-Book of Mental Diseases, p. 267.

Loss of the consensual movements of the pupil never occurs, except where the direct reflex movement is defective. Yet even then it may not be absent, or may merely have become sluggish. But it usually is wanting where the direct reflex has become absent in both eyes, as is not uncommon in the late stages. In cases in which the direct reflex is defective in one eye only, and while the consensual movements in it are likewise impaired, it has been observed by Redlich[1] that this eye is capable of giving rise to consensual reaction of the pupil of the healthy eye.

The Argyll-Robertson pupil is held by most authors to be present in but a small number of cases of general paralysis, and then usually in their later stages only; but the symptom certainly does occur in the initial stages of some cases of this disease, and an important inquiry would be to ascertain what the proportion of these cases is. (See also page 637 on the value of the Argyll-Robertson pupil.)

The pupil is sometimes irregular in shape, probably from the sphincter being more completely paralyzed at one part of its circumference than at another. Dawson and Rambaut found this sign present in so large a proportion of their cases as ninety-four per cent. As they found it also in cases of other sorts, they do not ascribe to it any great practical importance, though its absence would be, to some extent, against the diagnosis of general paralysis.

Oliver has studied the relation of the patellar-tendon reflex to the pupil-reflexes in general paralysis,[2] and has come to the following conclusions:

In some of the cases in the second stage of the disease, especially when the patellar-tendon reflexes were unequally exaggerated, there appeared to be an irregular and unequal spastic innervation of the two irides, causing irregularities in pin-point pupil forms.

In a few cases, especially in the third stage of the disorder, when the patellar-tendon reflexes were unequally diminished, the pupil of small size, and its shape somewhat irregular, the iris seemed to be acted upon but little by any powerful mydriatic.

In many cases, especially in comparatively young subjects in the third stage of the disease, when the patellar-tendon reflexes were unequally diminished, there appeared to be an unequal paralytic innervation of the two irides, the pupillary dilatation manifesting itself at times, though not, as a rule, in the eye with the greater amount of objective optic nerve-head degeneration and retinal change.

In a few cases, especially in men beyond middle life, in the third stage of the disorder, when the patellar-tendon reflexes were markedly diminished, and when the ataxies were quite pronounced, there were marked temporary asymmetries of pupillary form, one often being quite small and

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[1] Neurologisches Centralblatt, 1892, S. 80.

[2] Transactions of the American Ophthalmological Society, 1893.

irregular for several examinations, while its fellow was large and ovoid or oval.

In quite a number of cases, especially in the advanced stages of the disease, when the patellar-tendon reflexes were either unequally exaggerated or diminished, there was a failure of the irides to respond to even major degrees of light-stimulus; this not only being true for those subjects exhibiting a true spastic myosis, but also being more especially shown in those instances in which, with partial dilatation of the pupil, mydriatics failed to act.

In many instances, especially in the older cases, when the patellar-tendon reflexes were, as a rule, unequally diminished or even lost, not only was there failure of iris-response to the strongest light-stimulus carefully thrown upon the retina, but, when obtainable, the irides seemed to fail to react to the various coarse and rough subjective and objective procedures necessary to be used in order to evolve both separated and associated efforts for accommodation and associated efforts for convergence.

In some instances where ciliary muscle innervation could be satisfactorily obtained, both the spastic excitation and the paralytic enervation at times found by subjective reading-tests and objective study with the retinoscope appeared to be in direct ratio with the patellar-tendon reflexes.

In a number of cases where there was marked inequality of the pupils, with more or less want of reaction of the irides to light-stimulus, the patellar-tendon reflex on the side of the larger pupil seemed to be the more greatly diminished.

In several instances, especially during the very earliest stages of the disease, when the patellar-tendon reflexes were beginning to lessen to unequal degrees, there often appeared momentary secondary ataxic dilatation of the pupil during exposure to strong light (the paradoxical pupil-symptom).

In many cases, especially during the second stage of the disease, when the patellar-tendon reflexes began to become irregular and inconstant, pupillary inequalities, as expressive of unequal iris innervation and action, became more constant.

*Nuclear Paralysis.*—Paralysis of the orbital muscles is by no means so common a symptom in general paralysis as is the iridoplegia; but occasionally transitory paralysis of the third, fourth, and sixth nerves, accompanied by diplopia, is seen even in the prodromal and early stages of general paralysis. Ptosis also may be an early symptom, and transient nystagmus and twitchings of the eyelids. Very much rarer is permanent loss of power in one muscle, or the development of total ophthalmoplegia.

The various oculo-motor symptoms are caused by degenerative changes in the central gray matter of the aqueduct of Sylvius and fourth ventricle. Schütz[1] has found such changes, as have also Siemerling and Boediker,[2] and others.

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[1] Archiv für Psychiatrie und Nervenheilkunde, xxii.

[2] Ibidem, xxii. and xxix., 1897, p. 420.

*Optic Atrophy.*—Atrophy of the optic disk with its consequent amaurosis occurs in comparatively few cases of general paralysis: *e.g.*, in one thousand three hundred and eighty-six cases Hans Gudden[1] found it sixty-five times (four and nine-tenths per cent.), and in twenty-seven of these cases (two per cent.) there was defective knee-jerk. Uhthoff found atrophy in nine per cent. of cases examined by him. When atrophy does come on, it does not usually do so until a late period; yet cases have been recorded in which it was found at an early period. In one case it preceded any mental symptoms by two years, and in another by as much as eight years. Klein[2] and Uhthoff[3] describe a uniform, and more or less intense, opacity of the optic disk and retina, which extends far towards the periphery, and which is sometimes (Klein) accompanied by local dilatation of the vessels. More recent literature contains no further observations of this nature, and it is questionable whether the retinal opacity described is to be regarded as pathological. Hemianopsia has been found in general paralysis, and was probably due to softenings in the visual path or centre. Hirschberg[4] describes a case in which a permanent, and even progressive, central scotoma was an early symptom.

*Mind-Blindness.*—Besides its occurrence with focal cerebral lesions, mind-blindness is seen in cases of general paralysis, usually in the advanced stages. In one case of general paralysis under Wernicke's care,[5] which was examined by Schweigger,—where the symptom came on at an early stage, the patient's intelligence being still good,—the remarkable circumstance was noted that, with good acuteness of vision and without any absolute defect in the field, there were distributed over a great portion of the field a number of relative scotomata, within the area of any one of which, although objects could be seen by the patient, yet he could not tell what they were. The same objects in other parts of the field he was able not only to see, but also to identify.[6]

## MENINGITIS.

Meningitis in its various forms, both of the base and of the convexity, is apt to be associated with optic neuritis more or less well marked. In addition to this, when the meningitis is at the base of the brain, there are often symptoms derivable from the cerebral nerves,—ocular paralysis (page 602), pain or anæsthesia in the region supplied by the fifth nerve, defects in the field of vision from pressure on the optic tracts (page 572) or commissure (page 576), and so on.

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[1] Archiv für Psychiatrie und Nervenheilkunde, xxvi. 2, S. 430.
[2] Wiener medizinische Presse, 1897, Nr. 3.
[3] Bericht der ophthalmologischen Gesellschaft zu Heidelberg, 1883, S. 139.
[4] Centralblatt für Augenheilkunde, 1883, S. 27.
[5] Lehrbuch der Gehirnkrankheiten, iii. S. 552.
[6] An important study of the ocular symptoms in general paralysis is that by Oliver, Transactions of the American Ophthalmological Society, 1889.

*Acute Tubercular Meningitis.*—In acute tubercular meningitis there are found, probably in about fifteen per cent. of the cases, miliary tubercles in the chorioid. These tubercles appear as round, slightly prominent, pale yellowish spots, with softened margins, varying in size, as a rule, from 0.5 millimetre to 2.5 millimetres; situated in the neighborhood of the optic nerve and macula lutea; and unaccompanied by pigmentary changes. It is often found, on examination after death, that many more tubercles are present than those seen during life,—some having been too small to be recognized by the ophthalmoscope, while others lay too far towards the periphery of the fundus to be discovered. When chorioidal tubercles can be seen, the diagnosis of tubercular meningitis is determined, and the question which so often arises, Tubercular meningitis or typhoid? is answered. Unfortunately, the ophthalmoscope renders this important aid only occasionally, for it is only in some of the cases that tubercles are present in the chorioid at all. Again, although present, their small size may render it impossible to see them; and, finally, the difficulty of the examination is often insuperable where the patient is drowsy, and unable to control the constant involuntary rolling motion of the eyeballs. Miliary tubercles in the chorioid occur too, in cases of general acute miliary tuberculosis, in which there may happen to be no meningitis. Optic neuritis is more commonly present in this than in any other form of meningitis; also, in consequence of its tendency to attack the base of the brain, orbital paralyses are frequently seen.

*Cerebro-Spinal Meningitis.*—In cerebro-spinal meningitis, both in its sporadic and in its epidemic form, eye-symptoms of various kinds are extremely common at some stage of the disease. Swelling of the eyelids, conjunctivitis, and photophobia are frequent even in the early stages. The pupils may be contracted, or dilated, or unequal. There may be ulceration of the cornea, or parenchymatous keratitis, or deep purulent infiltrations of the cornea. Retinitis and plastic irido-chorioiditis, followed by retinal detachment, may be found, and purulent irido-chorioiditis with purulent infiltration of the vitreous humor, sometimes going on to the condition known as panophthalmitis, is also liable to appear. When the condition of the eye admits of an ophthalmoscopic examination, optic neuritis or neuro-retinitis will often be found present, or perhaps only congestion of the optic disk and engorgement of the retinal veins. Randolph,[1] in one of the thirty-five cases which he examined ophthalmoscopically, observed thrombosis of the central vein with hemorrhagic retinitis. Up to six days after the commencement of the illness the eyes were normal, but when Randolph examined them again, three weeks later, there were hemorrhages about the optic papillæ, the veins were enormously distended and tortuous, and the arteries were very much contracted.

Each epidemic of cerebro-spinal meningitis is apt to be associated with

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[1] Bulletin of the Johns Hopkins Hospital, iv., No. 32.

some one of the foregoing conditions as its special type of eye-affection, although conjunctivitis and pupillary anomalies tend to occur with every epidemic. The routine use of the ophthalmoscope in cases of cerebro-spinal meningitis, or where it is suspected, is important; for it will be remembered that the existence of good vision does not preclude the existence of optic neuritis. The presence of irido-chorioiditis, but more especially of optic neuritis, indicates a serious prognosis, and the discovery of optic neuritis may determine the diagnosis in cases where the general symptoms are indefinite or misleading.

In cerebro-spinal meningitis the prognosis for sight in cases which do not end fatally, and in which there is optic neuritis or irido-chorioiditis, is, for the most part, serious. Optic neuritis is liable to run into optic atrophy; and the exudation in the vitreous humor in irido-chorioiditis is apt to become organized and to shrink, causing retraction of the ciliary body and periphery of the iris and detachment of the retina. There is total posterior synechia, and through the contracted pupil the yellowish-white exudation in the vitreous may be seen. The eye becomes reduced in size and in tension. But it is possible for the optic neuritis to leave a healthy optic nerve behind it, and the irido-chorioiditis may be so modified in its violence, that the uveal tract and vitreous humor recover in such a way as ultimately to admit of good sight.

There are two views regarding the pathogenesis of the retinal and chorioidal affections in cerebro-spinal meningitis: 1. That the pneumococci which cause these inflammatory processes in the interior of the eye reach it by the lymph-spaces of the optic nerve from the inflamed cerebral meninges. Axenfeld[1] states that the pneumococci in this disease may fill the vaginal spaces of the optic nerve as far as the sclerotic without passing from here into the eye, and that it has not yet been proved that pyogenic organisms do reach the interior of the eye by this path. 2. That they reach it through the circulation as part of the general embolic poisoning of the system, and this mode of occurrence has been proved.

The micro-organisms may spread to the orbital connective tissue from the inflamed meninges through the sphenoidal fissure, or by way of the veins, and give rise to orbital cellulitis.

The inflammation of the optic nerve is either a descending neuritis, or is caused by the presence of pneumococci in the intervaginal lymph-space.

*Traumatic Meningitis.*—Falls and blows on the head, without fracture of the skull, are sometimes followed by optic neuritis, which may lead to optic atrophy with complete loss of sight, or may pass off without damage to vision. It is held that in these cases meningitis—preceded, it may be, by hemorrhages—has been set up by the blow, and that the inflammatory process, reaching the sheath of the optic nerve, creeps down it to the optic papilla.

[1] Archiv für Ophthalmologie, xl., 4, S. 184.

In some cases of hemorrhagic pachymeningitis, optic neuritis or congestion papilla has been seen, and has been found to be caused by hemorrhage into the sheath of the optic nerve. (See a'so page 582.)

### ACUTE HEMORRHAGIC ENCEPHALITIS.

As distinguished from inflammatory processes in the substance of the brain which may occur in connection with the various forms of meningitis, especially in the neighborhood of the primary inflammation, and also apart from the interstitial process which precedes the formation of a cerebral abscess, and from acute poliencephalitis superior (see page 591), we recognize a rare inflammatory process of the brain under the above title. It usually attacks circumscribed and often symmetrical districts of the cerebral substance, in which the vessels are much distended with blood, and where many minute hemorrhages, as well as inflammatory products, are present. The disease has been found as the result of epidemic influenza, and it has been looked on sometimes as an abortive form of cerebro-spinal meningitis, and, again, it has been seen in connection with ulcerative endocarditis.

In three cases of this affection Oppenheim[1] observed the presence of optic neuritis.

### HYDROCEPHALUS.

In congenital hydrocephalus, or in the hydrocephalus which appears in early infancy, congestion papilla or neuritic atrophy is sometimes seen; but, owing probably to the compensation provided for the increased intracranial pressure by the distensibility of the sutures, these changes in the fundus in these cases are not frequent. In the acquired hydrocephalus of later life, optic neuritis, or congestion papilla, passing over to optic atrophy, is the rule; and such cases may closely simulate an intra-cranial tumor in all their other symptoms as well. Bitemporal hemianopsia is apt to be present, owing to pressure exercised on the optic commissure by the distended floor of the third ventricle (page 578).

Leber records[2] a case in which the patient had been hydrocephalic from birth, and was liable to frequent and severe headaches, vertigo, occasional epileptic attacks, and marked loss of sight. At the age of twenty years the remarkable symptom of an almost continuous dropping of clear watery fluid from the nostril came on, associated with great relief from the headache, vertigo, and epileptic fits. Analysis of the fluid showed it to be identical in its composition with the cerebro-spinal fluid, and Leber believes that it actually was this fluid which flowed from the nostril. Cases of continuous dropping of watery fluid from the nostril with blindness due to optic neuritis, or neuritic atrophy, had already been published[3] by Nettleship (one case), and by Priestley Smith (two cases), and earlier cases

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[1] Lehrbuch der Nervenkrankheiten, Berlin, 1894, S. 518.
[2] Archiv für Ophthalmologie, xxix., 1, S. 278.
[3] The Ophthalmic Review, January, 1883.

had been made known by Elliotson,[1] Baxter,[2] and Paget,[3] while a more recent case is Emrys Jones,[4] whose patient was aged sixty-seven. The symptoms, more or less marked, were in general similar in all these cases, —namely, constant dropping of water from one nostril, severe headache, epileptic attacks, vomiting, stupidity, drowsiness, attacks of unconsciousness, delirium, and weakness in the legs, associated with a high degree of amblyopia or blindness due to optic neuritis, or to the resulting optic atrophy. Anosmia was present in some cases. When, occasionally, the dropping ceased, the head symptoms were aggravated. All the cases continued over a long course of years. The form of the skull left no doubt as to Leber's case being one of hydrocephalus. None of the other authors referred the symptoms in their cases to hydrocephalus, yet it now seems probable they were of this nature,—that they were, in short, cases of internal hydrocephalus coming on in adults, the ages of the patients which are given being twenty-two, twenty-three, twenty-eight, and sixty-seven. The composition of the fluid which came from the nostril was similar to that in Leber's case. The only cases among all these in which opportunity for post-mortem examination occurred were those of Paget and of Baxter, and in them there was no meningitis, and no focal disease within the cranium, and these seem to have been the main points to which at the autopsy attention was directed. In two of the cases that were merely clinically observed there was nasal polypus, and in one there were excoriations of the nasal mucous membrane, and the authors in these instances regarded these lesions as the origin of the watery discharge; but the peculiar nature of the latter, and the great frequency of nasal polypus, and other diseases of the nostrils, without any such discharge, render this view hardly tenable; and, indeed, these authors themselves have since then abandoned it. In the other cases the nasal passages were normal.

That this remarkable symptom may be connected with focal disease is shown by a case of tumor of the corpora quadrigemina, published by Nothnagel,[5] in which it was present. Here, too, analysis proved the fluid to be the cerebro-spinal fluid, and not an inflammatory product. At the post-mortem examination, along with the tumor of the corpora quadrigemina, there was found a large collection of fluid in the third ventricle, the lateral ventricles being empty.

Assuming, then, that the watery fluid which drops from the nostril in these cases is the cerebro-spinal fluid, the interesting question arises: How does it pass from the interior of the skull to the nostril? At neither of the post-mortem examinations which were made could any defect or abnormality of the bones at the base of the skull be found. Leber, however, is of

[1] The Medical Times and Gazette, September 19, 1857.
[2] Brain, January, 1882.
[3] Transactions of the Clinical Society of London, 1878.
[4] The Ophthalmic Review, vii. p. 97.
[5] Wiener medizinische Blätter, 1888, Nrs. 6–8.

opinion, that the defect might not be able to be seen by the naked eye, but that it is probably a very minute hole in the ethmoid bone, the result of the long-continued action of the increased intra-cranial pressure, and he suggests that, at the next opportunity, a colored fluid should be poured into the skull, in order to ascertain definitely the presence or otherwise of a communication between its cavity and the nostril. As a communication is present normally between the third ventricle and the subarachnoid space, it is not necessary to assume a direct opening of the third ventricle into the hypothetical fistula in the ethmoid. In Nothnagel's case it was held that the fluid did not come from the internal hydrocephalus, but rather from the subarachnoid space directly, and it was believed to have made its way into the nostril along the lymph-spaces of the olfactory nerve. Each olfactory nerve was atrophied through pressure.

### INFANTILE PARALYSIS.

Hemianopsia is a very rare symptom in this condition, but it has been recorded as occurring by Allen Starr[1] and by Henschen,[2] and in one case of the latter author's the hemianopic pupil was observed, but there was no autopsy. W. Koenig[3] records a case in which the hemianopsia was of transitory duration. In a child aged six and a half years, Sachs saw papillitis, blindness, left ptosis, sixth-nerve paralysis, right corneal anæsthesia, and right spastic hemiplegia, with a hemorrhagic cyst in the left temporo-sphenoidal lobe which pressed on the left crus cerebri.

### DEGENERATION OF THE CEREBRAL ARTERIES.

Retinal apoplexy in persons of advanced life may be taken as an indication of degeneration of the coats of the retinal vessels, and it is by no means an uncommon event to see hemiplegia following the retinal affection with an undefined interval of time. But short of retinal apoplexy there are ophthalmoscopic signs of changes in the arteries of the body generally and of the brain in particular, which have been made the subject of a careful and original study by Marcus Gunn.[4] He describes these signs as follows: The general reflex from the arteries is brighter than in the normal, and in particular the central light streak. This he holds to be due to a hyaline change in the arterial walls. The degenerated arteries are rigid, and consequently the circulation in the veins is impeded where they are crossed by arteries. As a result of this venous obstruction, there is set up an œdema of the retina, which may be either general or partial. The size of the arteries is not uniform, and a vessel may be widened or narrowed in a certain part of its course, this change being most often seen in the small

[1] The Medical Record, January 23, 1892.
[2] Loco citato, i. pp. 202, 205; ii. p. 362.
[3] Archiv für Psychiatrie und Nervenheilkunde, xxvii. (1895) S. 237.
[4] Transactions of the Ophthalmological Society of the United Kingdom, 1898.

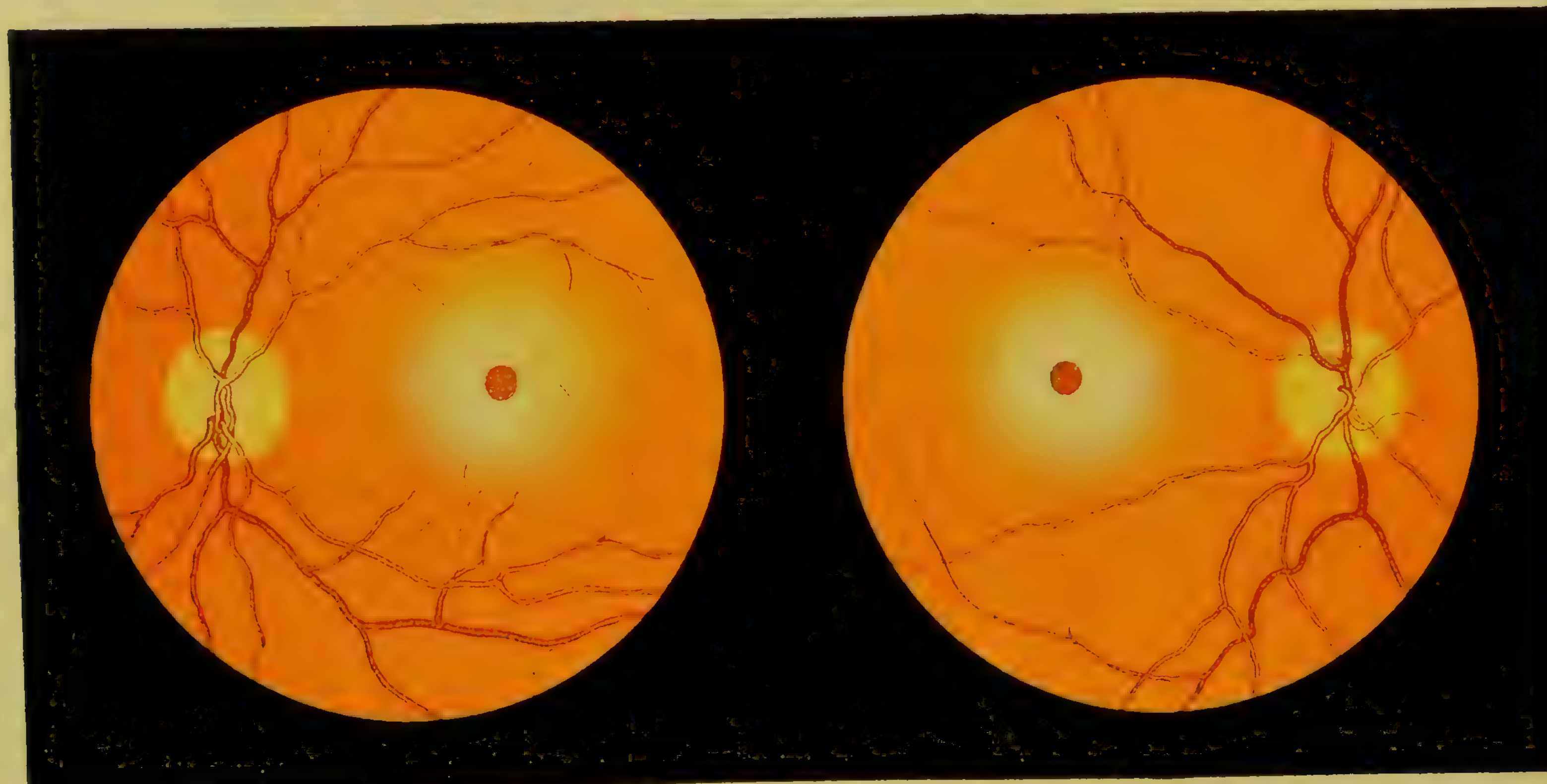
arteries in the region of the macula lutea. The arteries are sometimes very tortuous. The central streak is narrow, bright, and with spots of greater brilliance in it; a condition now and then seen in high hypermetropia, and after optic neuritis, in the vessels arising from the optic disk; but in the general disease vessels of the second and third magnitude are the ones chiefly affected. There is an early loss of translucency of the arteries, so that where a vein passes behind an artery the former cannot be seen.

On the other hand, if the vein covers the artery, the artery can be unduly seen through the blood column in the vein, because of the thickening of the arterial coat, and partial emptying of the vein by the thickened artery as the two cross each other. As a consequence of the hardness of the arteries, there is an interruption of the venous current, the vein is distended, and often hemorrhages take place along its course. The change in the arteries is a change in the coats, an irregular thickening; and with this there is a loss in carrying power, and hence tortuosity. The change in the veins is due to the damming back of the blood; the walls of the veins and capillaries undergo degeneration from the œdema induced, and hence hemorrhages in the retina arise. The changes usually first occur between forty and fifty, and if well marked at this age the prognosis is grave, from the liability to thrombosis of cerebral arteries and cerebral hemorrhage.

The subjects of these vascular changes are often liable to migraine, indigestion, or gout. Chronic alcoholism is also a potent factor in the causation. This affection of the vessels is in close association with renal disease, but the vessels of the eye and brain may be affected before the kidney. Gunn examined the eyes of all the hemiplegic patients who were in the National Hospital at one time. Out of seventeen cases, ten showed the above changes, while most of the remaining seven cases had cardiac or aortic disease, making it probable that the causation was embolic.

*Amaurotic Family Idiocy* (Sachs), *or Infantile Cerebral Degeneration with Symmetrical Changes at the Macula Lutea* (Kingdon and Risien Russell).—This remarkable disease occurs in children in the first year of life. At birth the infants are sound and healthy, and continue to thrive both mentally and physically in a normal way for a few months. A cessation in their mental development is then noticed, and marked idiocy gradually becomes established. Along with this, paresis or paralysis of the greater part of the body appears, and it may be flaccid or spastic, while the reflexes may be deficient or increased. Hyperacusis is often present. A diminution in vision, terminating in absolute blindness, with certain characteristic ophthalmoscopic appearances, is one of the chief and earliest symptoms of the disease, and nystagmus and strabismus are sometimes present. A slowly increasing marasmus leads to a fatal termination at about the age of two years, as a rule. In one case, when reported, the patient was four years old. The disease is very liable to attack several children of the same fam-

PLATE I.



Disease in the region of the yellow spot in each eye in an infant. (Waren Tay.)

ily. It was Waren Tay who first observed and described[1] the peculiar ophthalmoscopic appearances in these cases, while Sachs subsequently described[2] the clinical history, the general symptoms, and the morbid changes in the brain. Neither author at first recognized the interdependence of the ocular disease with that of the general nervous system. Kingdon and Risien Russell[3] and Hirsch and Holden[4] have added much to our knowledge of the pathology of the disease. The ophthalmoscopic appearances are as follows: The optic disks in the early stages are healthy. In the region of the macula lutea of each eye there is a large white spot, tolerably diffuse, with softened edges, which covers a space about twice the size of the optic papilla. At its centre there is a brownish-red, fairly circular spot, contrasting strongly with the white patch surrounding it. This central spot, Tay says, does not look like a hemorrhage, nor as if due to pigment, but seems to be a gap in the white patch, through which healthy structures are seen. In fact, the appearance may most suitably be compared with that which we are familiar with at the yellow spot in cases of embolism of the central artery of the retina. This appearance is represented in the accompanying illustration (Plate I.), taken from Mr. Tay's original drawing, which he has kindly lent the writer for this chapter. At a later period, with complete amaurosis, atrophy of the optic nerve, in addition to the appearances at the yellow spot just described, is found. Precisely the same appearances were present in all the cases afterwards observed in which the ophthalmoscope was used.

As regards the diseased appearances in the brain, in the six cases, out of a total of twenty-four observed, in which the brain was examined,—viz., two by Sachs, three by Kingdon and Risien Russell, and one by Hirsch and Holden, the diseased appearances were similar. Sachs refers these appearances to an arrest of development, but the other authors regard them as degenerative changes. Hirsch, moreover, found that the diseased changes were present not merely in the cerebral cortex, but that they extended to the nerve-cells of the entire nervous system, and he suspects that the disease is due to a toxic influence, although, unless it be in the mother's milk, he is unable to suggest the origin or nature of the poison. The essential change is one of degeneration of the pyramidal cells of the cerebral cortex. These cells are found in all stages of degeneration, from a comparatively slight one to one so pronounced that, as seen in specimens prepared by the silver method, a black, irregular mass is the sole representative of what was once a cell. In the pons and medulla oblongata degeneration of the pyramidal fibres and of the fillet has been found; and in the spinal cord well-marked degenera-

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[1] Transactions of the Ophthalmological Society of the United Kingdom, 1881, p. 55, and 1884, p. 158.

[2] The New York Medical Journal, May 30, 1896; The Journal of Nervous and Mental Disease, 1887, p. 541.

[3] Transactions of the Medico-Chirurgical Society, lxxx., 1897.

[4] The Journal of Nervous and Mental Disease (New York), July, 1898, pp. 538–555.

tion of both the crossed and direct pyramidal tracts was seen (Kingdon and Risien Russell). A microscopical examination (Treacher Collins) of the eyes in one case showed that the retina at the yellow spot was much thickened, owing to enlargement of the outer molecular layer, the tissue of which was much spaced out, cavities being left here and there. The condition was apparently due to œdema. The other layers showed no change, and elsewhere than at the yellow spot the retina was healthy. The optic nerves were atrophied, but there was no microscopical sign of former neuritis. Holden ascertained that the changes in the retina consisted in diseases of the ganglion cells similar to that which is found in the cerebral cortex. The fact that the ganglion-cell layer is absent at the fovea centralis and is thickest in the immediate neighborhood of the latter all round, explains the peculiar ophthalmoscopic picture.

As regards the etiology of the disease, family predisposition has been strongly marked, inasmuch as the twenty-three cases observed belonged to eleven families, as many as five cases having been seen in one family of seven children. Among the causes which could be assigned were marked neurotic taint, blood relationship between the parents, and traumatism of the mother during pregnancy. Most, if not all, of the cases occurred among Jews. Syphilis was not assigned as a cause in any of the cases, and its absence is distinctly noted in seven of the eleven families.[1]

*Paralytic Vertigo.*—In this curious complaint, which was first described by Gerlier,[2] and which has, so far, been observed only in the neighborhood of Geneva, certain eye-symptoms play an important part in the general picture of the disease, the classic symptoms of which are pains in the nape of the neck, troubled vision with ptosis, bending of the head forward on the breast, occasionally paresis of the lower limbs, and vertigo, all coming on in attacks which last for some hours.

Ptosis is the only objective symptom met with in examination of the eyes, and it may last after the others have disappeared. The eye-symptoms are mainly subjective in character,—viz., cloudiness of sight at the beginning of an attack; double vision, sparks, oscillation, dancing of objects before the eyes. Gerlier states that the eye has an undoubted influence in causing an attack; the bustle of people in towns, the flowing of water

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[1] In addition to the references already given there are the following: H. Magnus, Klinische Monatsblätter für Augenheilkunde, 1885, S. 42; Goldzieher, Wiener medizinische Wochenschrift, 1885, Nr. 11; Knapp, Bericht der ophthalmologischen Gesellschaft zu Heidelberg, 1885, S. 217, and Bericht der International Ophthalmologischen Congress zu Heidelberg, 1888; Hirschberg, Centralblatt für praktische Augenheilkunde, 1888, S. 15; Wadsworth, Transactions of the American Ophthalmological Society, 1887, p. 572; Kingdon, Transactions of the Ophthalmological Society of the United Kingdom, xii. p. 126, xiv. p. 129; Carter, Archives of Ophthalmology, January-April, 1893; Koller, Transactions of the American Ophthalmological Society, 1896, p. 661; Peterson, The Journal of Nervous and Mental Disease (New York), July, 1897, p. 529 (but it is not clear that this case was a true one of the disease).

[2] Revue médicale de la Suisse Romande, January, 1887; Brain, Winter Number, 1889.

under bridges, the glare of strong light, the gazing at any large open space, is enough to bring on an attack of vertigo. We have to deal with a psychic impression whose source is the sight.

Eperon [1] noted marked hyperæmia of the papilla in two cases; indeed, in one of them he thought there was papillitis with peripapillary hemorrhages; and he is inclined to believe that the disease consists in hyperæmia of the meninges of the brain, with consecutive œdema. Sulzer [2] found similar ophthalmoscopic appearances, with peripheral defects in the fields of vision.

### PARALYSIS AGITANS, OR PARKINSON'S DISEASE.

In some cases of this disease a fine vibratory tremor may be noticed along the margin of the upper lid, especially when the eyelids are closed; and on an attempt at passive opening of the lids they will be found unusually rigid. The slowness of muscular action, usually conspicuous in paralysis agitans in all movements, rarely, as Gowers says, affects the muscles of the eyes. If the patient is told to look in any given direction, the eyes are instantly turned, while the head slowly follows them; yet, according to Koenig,[3] the amplitude of the ocular movements is reduced, and there is sometimes a lateral nystagmus. The pupils are generally normal, the myosis present being probably senile myosis. Koenig has noticed spasm of accommodation in several cases, and in two cases he observed transitory amaurosis.

### ENCEPHALOPATHIA SATURNINA.

Even in the less severe cases of this affection, transient hemianopsia or amaurosis, lasting for hours, may be met with. There need not be any renal disease, and the defect of sight must be taken as being due to the effect of the lead-poison on the brain. In those cases in which acute cerebral disturbance (convulsion, delirium, or coma) sets in, it is often attended by optic neuritis, in which the swelling is considerable and accompanied by hemorrhages.[4] The neuritis may pass off under treatment without impairment to sight, or consecutive atrophy may come on. Occasionally the fields will be found contracted, as in hysteria, without any ophthalmoscopic appearance. The pupils are sometimes unequal.

Inasmuch as the symptoms of bad cases of lead encephalopathy usually consist of severe headache, vomiting, and convulsions, the spasm being in some cases epileptiform, it is evident that, when intense double optic neuritis is added, the diagnosis between this disease and cerebral tumor has to be considered. B. Bramwell, who has extensive experience of lead encephalopathy, says, "So closely do the two conditions [tumor and lead enceph-

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[1] Revue médicale de la Suisse Romande, 1889, No. 1.
[2] Ibidem, 1893, No. 11.
[3] Annales d'Oculistique, Juillet, 1893.
[4] Gowers, Diseases of the Nervous System, 2d ed., i. p. 955.

alopathy] in some cases resemble each other, that I never commit myself to a positive diagnosis of intra-cranial tumor without having previously excluded lead-poisoning." [1]

The aids in the diagnosis will, of course, be the presence or absence of the characteristic blue line on the gums, as also of anæmia, colic, constipation, wrist-drop, and lead in the urine; together with the previous history and the patient's occupation.

## EPILEPSY.

In idiopathic epilepsy a visual aura is more common than any other special-sense aura. It often consists in subjective sensations of light, color, flames, megalopsia and micropsia, etc., and also in visual hallucinations, such as an old woman, a soldier, faces, beautiful scenes, etc., or in simple homonymous hemianopsia. In epilepsy due to organic brain-disease a visual aura occurring always in homonymous sides of the fields is important, as indicating the region of the brain (occipital lobe) in which the discharge originates. By electrical irritation of the occipital lobe in dogs, epileptic fits may be induced,[2] but it is well understood that the posterior areas of the brain merely possess the power of originating the discharge of energy and of transferring it to the motor area. If the latter be extirpated, no epileptic fit can be produced by irritation of the cortical centre for vision.[3] Since cortical epilepsy beginning with hemiopic visual aura is a condition in which operative interference may be undertaken, our knowledge of the seat of the centre for vision is, here, not without practical importance.

At the onset of a fit there is very often conjugate lateral deviation of the eyes to the opposite side from the side of the body on which the convulsions commence, with rotation of the head in the same direction. This may continue during the whole of the spasmodic stage, or the eyes may suddenly be turned in the opposite direction,—a symptom, probably, of fatigue in the centre for this motion. Sometimes, associated rolling motions of the eyes may be seen, or nystagmus. Both orbicular muscles, like the muscles of deglutition, the diaphragm, etc., being innervated from each side of the brain, take part in the spasm, even when only one side of the body is affected.

The condition of the pupils is variable, often even in one and the same fit. At the onset the pupils are usually normal or contracted, but during the tonic spasm they become dilated, and remain so until consciousness returns. The light-reflex is lost,—a point of importance in the diagnosis of

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[1] Intra-Cranial Tumors, p. 128.

[2] Rosenbach, Archiv für pathologische Anatomie und Physiologie, xcvii. 3, and Neurologisches Centralblatt, 1889, Nr. 9; Unverricht, Archiv für klinische Medicin, xliv. 1; and many other authors.

[3] Yet, as Rosenbach observed, after extirpation of the Rolandic area, conjugate lateral motions of the eyes could be caused by irritation of the occipital lobe as readily as before.

a true epileptic fit from an hysterical attack, in which latter it is retained. After the fit, rapid variations in the size of the pupil (hippus) may sometimes be seen, and are valuable as evidence of the fit having been a genuine one.

The ophthalmoscopic appearances during a fit are not very easily observed, but they have been studied by several observers, and would seem to vary in different cases. Sometimes they are normal; sometimes there are marked pallor of the optic papilla and contraction of the blood-vessels; and, again, in other cases there are hyperæmia of the papilla and engorgement of the retinal veins. Optic neuritis and optic atrophy, which have been noted in some cases of epilepsy, are to be regarded as complications which have nothing to do with the epilepsy as such. In the intervals between the attacks the fundus may be normal, but a very usual condition is a high degree of hyperæmia of the retina and optic papilla, which may last for some hours or for days, or may become chronic.

After the fit, and in some cases as a chronic condition, in like manner as in some other neurasthenic states, the fields of vision are found to be contracted concentrically, and the boundaries for color may also be contracted, but without transposition or reversion (Oliver)[1]; or there may be color-blindness, and the central acuteness of vision may be reduced. The contracted condition of the fields is another valuable aid in the detection of simulation.

Transitory amblyopia, in the widest sense of the term (migraine, scotoma, etc.), is more frequent in connection with epilepsy than under any other condition. It may precede the true epileptic attacks by many years, or it may occur along with, or for an hour or so before, the fits, or it may be substituted for them. The circumstances that this transitory blindness is often accompanied by disturbances in the speech, in the intelligence, or by passing paralysis, and that both eyes are usually attacked by it, frequently in the form of homonymous hemianopsia, render it evident that its cause resides in the visual cortex. Yet the amblyopia is in some few cases monocular, and must then be referred to disturbance in the circulation of the optic nerve or the retina.

The question raised by Wigglesworth and Bickerton,[2] concerning the connection between epilepsy and errors of refraction, is one which hardly comes within the scope of this article, yet it may be thought desirable that some reference should be made to it here. Those authors, basing their opinion on the examination of one hundred cases of epilepsy, have arrived at the conclusions that, given a predisposition to epilepsy, an abnormal condition of the refractive media of the eye may at times prove the exciting cause of the disease, and that the cases examined seem to justify the assumption that certain cases of epilepsy do occur in which the attack is

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[1] Journal of the American Medical Association, September 26, 1891.

[2] Brain, January, 1889.

induced by the undue strain put upon the muscular apparatus of the eye by reason of an abnormality of refraction. This being so, they believe that, if the refractive error be corrected with glasses, there is reason to anticipate that the fits will subside, provided this treatment be resorted to at a sufficiently early period of the case. Work Dodd, in an admirable paper,[1] founded on the painstaking examination of one hundred epileptics, comes to a very similar conclusion. He states that errors of refraction may excite epilepsy; that the correction of errors of refraction will, in combination with other treatment, in many cases cure or relieve the epileptic condition; that in his one hundred cases forty-nine patients were so cured or relieved; and that in some cases, when the refractive error has been corrected, the epilepsy will continue, generally in a modified form, in consequence of other irritation, even though the error of refraction may have been the exciting cause of the fits in the first instance. Such careful and laborious studies cannot be regarded as other than worthy of consideration, and as prompting to further investigations on the subject, especially as concerns the permanence or otherwise of the cures.

Stevens, of New York,[2] has also formed the opinion that refractive errors are causes of epilepsy. This author holds, too, that certain anomalous conditions—insufficiencies—of the orbital muscles may be productive of epilepsy, a view which has met with considerable adverse criticism.

## CHOREA.

The theory put forward by Hughlings Jackson that chorea is the result of cerebral embolism is now, according to Gowers,[3] merely of historical interest, and quite untenable. Nevertheless, it is worthy of note that in a case of the writer[4] embolism of the central retinal artery of the left eye, and chorea, chiefly of the left side of the body, came on simultaneously, and that in a case of A. Benson[5] three attacks of chorea had preceded a retinal embolism. In Benson's case the patient had had acute rheumatism, but there was no cardiac disease; in the writer's case there had not been rheumatism, and the heart was healthy. Again, Leber mentions[6] a case of chorea in which sudden blindness of one eye came on, and in which, some months later, he found optic atrophy of, he thinks, embolic origin. In this case there was an aortic murmur. Possibly, therefore, the embolic theory of chorea may yet be found to be more tenable, at least for some cases of that affection, than Gowers believes.

In chorea the head and eyes may participate in the irregular movements, being moved to one side by the consentaneous spasm of the quick,

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[1] Brain, Winter Number, 1893.
[2] Functional Nervous Diseases, their Causes and their Treatment.
[3] Diseases of the Nervous System, 2d ed., ii. p. 616.
[4] Royal London Ophthalmic Hospital Reports, viii. p. 181.
[5] The Ophthalmic Review, 1886, p. 1.
[6] Handbuch der Gesammten Augenheilkunde, v. S. 870.

jerky form characteristic of chorea; and Gowers specially directs attention[1] to the point, that this spasm may be so unequal in the two eyes as to cause brief diplopia, although it is insufficient to produce a visible variance of the ocular axes. The point is of diagnostic importance, as was shown in a case of Gowers's. The patient had choreic movements, with optic neuritis and headache. The two latter symptoms suggested that the movements might be symptomatic of focal brain-disease, and therefore not true chorea. This doubt was increased when the patient shortly afterwards complained of occasional double vision, although no defective movement of the eyes could be detected. The case ran the ordinary course of chorea, and made a good recovery. Gowers finds that diplopia in patients suffering from chorea is by no means infrequent, although, not being constant, little attention is paid to it, and it is rarely, if ever, mentioned spontaneously.

## DEFORMITIES OF THE SKULL.

Along with certain congenital deformities of the skull, optic neuritis, or post-neuritic optic atrophy, is seen.

The deformity most commonly attended by these serious eye-complications is a high, narrow, and long skull, which looks as though it had been subjected to great lateral pressure, and is known as oxycephaly. At the anterior fontanelle and along the sagittal suture a prominent ridge of bone is present. According to Virchow, the deformity is caused by premature synostosis of the sagittal suture, which may be, and often is, accompanied by inflammatory intra-cranial processes, mainly pachymeningitis.

The orbits in these skulls are often very shallow, and as a result of this the eyeballs are protruded, and at first may be mistaken for myopic globes. The orbits, too, are lofty in proportion to their width. The vision is defective in greater or less degree from birth, some of the patients being born almost, or quite, blind of both eyes, while others enjoy fair vision in one or both eyes during the early years of life, but the sight gradually diminishes until, as a rule, before adolescence, absolute amaurosis is reached. In some patients of advanced life, however, very imperfect vision has been retained.

The ophthalmoscope discovers the presence either of optic neuritis, which is sometimes of the choked-disk type, or of post-neuritic atrophy, more or less advanced. The usually accepted view as to the cause of the optic-nerve affection is, that it is due to the meningitis. Michel thought it might be caused, or at least promoted, by compression of the optic nerve in the optic foramen, which is sometimes found of abnormally small size in these skulls. Indeed, in a case of which Manz obtained a post-mortem examination, the optic nerves presented a marked constriction where they had passed through

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[1] Transactions of the Ophthalmological Society of the United Kingdom, iv. p. 306.

the optic foramina. In one case (Stood) the ophthalmoscopic appearances were normal, although amaurosis was present; and here the author assumed as the cause of the blindness defective development of the occipital lobes, the skull being high and narrow and, in this instance, short in its antero-posterior measurement.

The patients are often mentally defective, or subject to convulsions or to epileptic fits, and frequently die young. Friedenwald asks whether there may not be an indication for relief of intra-cranial pressure by trephining in these cases.[1]

A remarkable case of congenital deformity of the skull with extreme proptosis of each eyeball, which belongs to this category, has been published by Henry Power.[2] The subject of it was an infant born after an easy delivery at full term, and which died on the thirtieth day. The case affords a valuable instance of the effect of premature synostosis in producing such deformities, and their resulting displacements of the globes. Plate II., Fig. 1, represents the appearances during life. The eyeballs projected so far that the margins of the lids were behind the vertical transverse equators, and the dislocated globes could not be reduced into their sockets. The corneæ were hazy and threatened ulceration, so that no ophthalmoscopic examination was possible. The head was very short in the antero-posterior diameter, but of great vertical height, especially in the frontal region, where it became cone-shaped. There was not any ridge of bone on the vertex. After hardening in spirit, a sagittal section of the head was made, passing through the vertical meridian of the right cornea. The right orbit, which was thereby opened, was found to be extremely shallow, its depth being only 2.5 centimetres from its inferior border to the optic foramen. The height of the orbit, on the other hand, was increased, measuring as it did thirty-two millimetres at its most anterior part, while twenty millimetres is the normal measurement at this period.

Owing to the prolongation upward of the head into a blunt cone, the form of the brain and the convoluted pattern of the cerebral hemispheres were greatly altered. The corpus callosum was absent, and, as is often noticed in connection with this defect, the surface of the cerebrum presented a microgyrous condition in certain localities. This was particularly noticeable in the frontal region.

Plate II., Fig. 2, from a photograph taken by Professor D. J. Cunningham, of Trinity College, Dublin, represents the appearances of the interior of the cranium after removal of the brain. The floor of the orbit was

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[1] Michel, Archiv für Augenheilkunde, xiv.; Archiv für Ophthalmologie, xii., 2, S. 133; Hirschberg, Centralblatt für praktische Augenheilkunde, 1883, S. 1; Stood, Klinische Monatsblätter für Augenheilkunde, 1884, S. 248; Manz, Bericht der ophthalmologischen Gesellschaft zu Heidelberg, 1887, S. 18; Meynerz, Deutsche medicinische Wochenschrift, 15 März, 1888; Weiss und Brugger, Archiv für Ophthalmologie, xxiv., 1, S. 55; Friedenwald, The American Journal of the Medical Sciences, May, 1893.

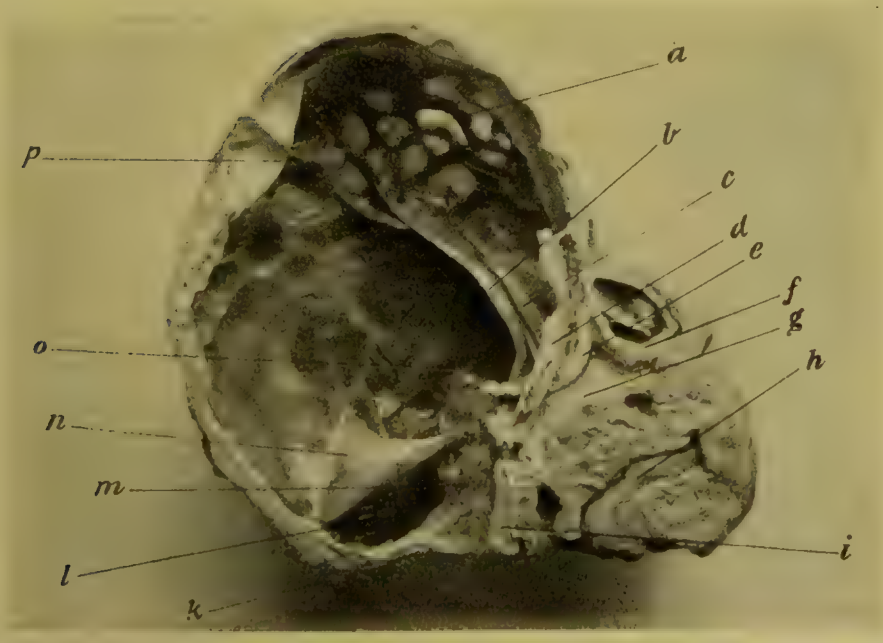
[2] Transactions of the Ophthalmological Society of the United Kingdom, 1894, xiv. p. 212.

PLATE II

FIG. 1.

Appearances of Mr. Power's case during life.

FIG. 2.




Appearances of interior of cranium in Mr. Power's case.—*a*, pits in the cranial vault in which the small frontal convolutions were placed,—thinning of the bone; *b*, sharp edge which in adult skull is formed by posterior border of lesser wing of sphenoid; *c*, roof of left orbit; *d*, roof of right orbit in section; *e*, optic nerve; *f*, sclerotic; *g*, nasal fossa; *h*, tongue; *i*, basi-occipital; *k*, foramen magnum; *l*, posterior cranial fossa; *m*, petro-mastoid; *n*, tentorium; *o*, middle cranial fossa; *p*, falx cerebri cut short.

horizontal, but the roof was almost vertical in position, being directed upward and forward at an angle of forty-five degrees, and becoming continuous with the vertical plate of the frontal bone, into which it passed with only a small projection to mark the orbital edge. A perpendicular line from the front of the vertical portion of the frontal bone would fall entirely behind the globe. The optic nerve from its entrance at the optic foramen ascended sharply, and remained in close relation with the displaced roof of the orbit, till it bent downward to enter the sclerotic. A careful examination, to which the skull was submitted by Professor Cunningham,[1] displayed the cause of these remarkable distortions. It was ascertained that, with the single exception of the joint between the ex-occipital and the basi-occipital, every suture and synchondrodial joint in the skull was firmly ossified. The consequence of the solidification of the base and the ossification of the vault into a continuous bony sheet was the general uplifting of the front part of the roof of the cranium by the growing brain, and the simultaneous dragging up with it of the orbital plates, to which latter is due the extreme shallowness of the eye-sockets. It was also found that the convolutions of the brain had pressed so hard against the ossifying cranial vault, that the normal dimples on its inner surface had become deep bony pits, like a honeycomb, or like the cells in the stomach of a camel, and the floors of these pits had been made exceedingly thin and diaphanous.

Another form of skull which has been observed by Vossius[2] to be accompanied by optic neuritis or optic atrophy is one in which the head is too long antero-posteriorly and too short in its vertical measurement. There is no hydrocephalic enlargement, and the face is almost square.

Again, Oppenheim[3] has seen cases in which there was abnormal width of the skull with protrusion of the zygomata, while in other diameters it was of normal dimensions; and here, too, optic neuritis passing over to atrophy was noted.

The simple microcephalic skull is also liable to be complicated with amaurosis.

## DISEASES AND INJURIES OF THE SPINAL CORD.

The organic diseases of the spinal cord, and those which, for the present, are held to be such, and in which eye-symptoms occur, are the following: tabes dorsalis; hereditary ataxy, or Friedreich's disease; myelitis; syringomyelia; Morvan's disease; myotonia congenita, or Thomsen's disease; acute ascending paralysis, or Landry's disease. Disseminated sclerosis and cerebro-spinal meningitis have been treated of under the head of Diffuse Diseases of the Brain.

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[1] The writer was indebted to the late Dr. Kanthack, at that time curator of the museum at St. Bartholomew's Hospital, where the specimen had been deposited, for his kindness in lending it to him for this further examination.

[2] Klinische Monatsblätter für Augenheilkunde, 1884, S. 172.

[3] Lehrbuch der Nervenkrankheiten, S. 560.

### TABES DORSALIS.

The eye-symptoms which may be found in this disease are: 1. Atrophy of the optic nerve. 2. Paralyses and ataxy of orbital muscles. 3. Pupillary alterations. 4. Paralysis of accommodation. 5. Narrowing of the palpebral fissure. 6. Imperfect closure of the eyelids, associated with twitchings in the orbicularis palpebrarum. 7. Epiphora. 8. Reduction of the intra-ocular tension.

*Atrophy of the Optic Nerve.*—This is one of the most grave symptoms of this serious disease, and occurs in about twenty per cent. of the cases. Its onset is more frequently in the pre-ataxic period than subsequently. It may even be the very first symptom, and occasionally precedes lightning pain, loss of knee-jerk, and other spinal symptoms by a long interval of time,—from two to twenty years. But optic atrophy does sometimes commence in the later stages of locomotor ataxy.

Coming on in the pre-ataxic stage, optic atrophy seems very often to have—as Benedikt first pointed out,[1] and as has since been noted by many observers—a favorable influence on the spinal disease, symptoms of tabes already existing being ameliorated or disappearing, while the further progress of the disease seems to be retarded or averted. Indeed, it appears to be rare for a tabetic patient who goes blind at an early stage of the disease to become ataxic later, while the lightning and other pains become less severe after amaurosis is established. But if the ataxy be well marked, it does not improve with a subsequent development of optic atrophy.

Usually both eyes are affected with optic atrophy simultaneously or with a very short interval, but sometimes the onset in one eye precedes that in the other by a long time. Leber saw the interval as much as four years[2] in one case. At the onset of the optic-nerve affection patients often have some photophobia and subjective sensations of sparks and colored lights and *mouches volantes*, and complain of a fog or smoke between their eyes and the objects they look at, and in two cases Berger[3] noted erythropsia. The interval between the commencement of the optic-nerve disease and the advent of complete blindness varies considerably in different cases. Probably one year is an average interval; but it is often only a few months, and has been in some cases as long as fifteen or seventeen years, and in the slower cases there may, for months at a time, be no progress in the blindness. Martin[4] points out the remarkable fact that Romberg's symptom is not generally found in blind tabetics.

Color-blindness is almost always associated with the optic atrophy, but need not be in direct proportion to the diminution in the acuteness of vision,

[1] Wiener medizinische Presse, 1881, Nrs. 1, 2, 4, 5.
[2] Handbuch der Gesammten Augenheilkunde, v. S. 866.
[3] Archiv für Augenheilkunde, xix. S. 408.
[4] Brain, 1890, p. 413.

nor to the contraction of the field. Indeed, occasionally, before the ophthalmoscope can detect any change in the papilla, there is a derangement of the color-sense. This will be associated with defective sensitiveness for the light difference, while in congenital color-blindness the light-difference will be normal. Green is the color which is first lost.

The field of vision becomes contracted, but there is no one form of contraction characteristic of the disease. The contraction may be, and perhaps most frequently is, concentric; or it may begin on the temporal side of the field only, or on the nasal side, or above, or more rarely below, and then subsequently start in the other parts of the periphery. The concentric form of contraction commonly commences by sending re-entering angles of blindness into the field, which cut across the boundaries for the color-fields. But the color-fields themselves, too, become contracted, and that sometimes before the field for white is affected: so that in doubtful cases an examination of the color-fields may help in clearing up the diagnosis. Mental worry may cause temporary functional increase in the contraction of the fields in tabes. Central scotoma, if it occur at all in tabes, as such, does so with extreme rarity.

In respect of the ophthalmoscopic appearances, there is a want of accord among authors as to whether a short period of hyperæmia precedes the atrophic appearances. Most writers make no statement on the point, others deny any such hyperæmic stage, and a few maintain its occurrence. The truth is, the opportunities for studying this stage are not frequent. In commencing atrophy, the nasal side of the papilla, which is normally pinker than the temporal side, becomes gray, and gradually the lamina cribrosa comes into view, atrophic cupping is developed, and the whole papilla presents a uniform grayish hue. Subsequently, when the case is of some standing, according as connective tissue is developed in the lamina cribrosa, the grayish hue is changed to a glistening white and the retinal arteries and veins are reduced to the finest threads. Leber has shown that the atrophic process advances from the periphery towards the axis of the optic nerve; and it is not a descending atrophy from the brain, but one which begins in the retro-bulbar part of the optic nerve.[1]

The treatment of optic-nerve atrophy is about as hopeless as that of any disease well can be. Various remedies have been tried for it. Benedikt[2] states that, unless in those cases which are indubitably on a syphilitic basis, mercury and iodide of potassium are likely to be not only useless, but even very injurious, by hastening the progress of the blindness. Leber holds a similar opinion, but goes further, in that he believes mercury to be mischievous even in syphilitic cases. Iodide of potassium, arsenic, iron, cyanide of gold (Galezowski), salicylate of sodium with Turkish baths, strychnine hypodermically, inhalations of nitrite of amyl, and suspension have been praised, each in its turn, and have each failed to gain more than

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[1] Archiv für Ophthalmologie, xiv., 2, S. 177. [2] Ibidem, xliii., 1897, p. 701.

a temporary reputation. In forming an opinion as to the efficacy of a remedy in this disease it must be borne in mind, that, as already stated, even without treatment, periods often occur in which no advance in the blindness takes place, or in which the patient may think his sight to be somewhat better.

The relation between the optic atrophy and the spinal disease is not well understood; but the former, probably, is merely a manifestation of a diseased process in the optic nerve, similar to that which attacks the posterior columns of the cord. It cannot be the result of some influence which the spinal cord has over the nutrition of the optic nerve, for the latter may become atrophied long before it can be assumed that spinal disease is present. That it is not a direct continuation of the disease from the spinal cord is evident, both from the investigations of Leber above referred to, and because the spinal degeneration usually ceases below the medulla, or at most it affects the nerve nuclei in the latter. Wharton Jones propounded the view,[1] that the optic-nerve disease results from an affection of the sympathetic by paralysis of the vaso-motor nerves, and consequent hyperæmia of the optic nerve, followed by atrophy of it; and recently Berger[2] has put forward a similar view.

*Paralyses and Ataxy of Orbital Muscles* (see also page 590).—Paralyses of orbital muscles in tabes are not uncommon, occurring in about twenty-five or thirty per cent. of the cases. By far the greater portion of these paralyses appear in the pre-ataxic stage, and even as an initial symptom. There are two chief forms of them,—the transient paralysis, which lasts a few days or weeks, and may recur, and the permanent paralysis of one or two muscles. The diplopia produced by these paralyses is often the symptom which first induces the patient to see his doctor. Any orbital nerve may be affected, but the sixth nerve seems to be the one most commonly paralyzed. The third nerve also is often paralyzed, and the levator palpebræ is frequently involved, with resulting ptosis. De Watteville[3] and others have found loss of power of convergence (difficulty of fusion) not rarely present in beginning tabes, and double exterior ophthalmoplegia, as well as double sixth-nerve paralysis, is sometimes seen. There can be no doubt that such conditions as these three last mentioned have a nuclear disease as their cause, and it is probable that some of the other oculo-motor disturbances in tabes are of nuclear origin. Some of them, on the other hand, may be due to peripheral neuritis, and the orbital nerves may undergo atrophy without their nuclei being altered.

Ocular ataxy (saccaded movements of the eyeballs, ocular myoscismia) is another but less common symptom. It is sometimes erroneously called nystagmus; but nystagmus is a constant oscillatory motion of the eyeballs, both while the eyes are at rest and when an object is fixed, and is extremely

[1] British Medical Journal, July 24, 1869.

[2] Loco citato.

[3] Neurologisches Centralblatt, 1887, Nr. 10.

rare in tabes. In ocular ataxy, so long as the eyes are at rest there is no oscillation or twitching; but as soon as an object is carefully looked at, and especially if followed when in motion, and more particularly at the end of the latter, a slight twitching of the eyeballs is seen. It is liable to be found in every stage of tabes.

*Pupillary Alterations.*—Myosis is the usual state of the pupil in tabes, and it is to be regarded as of the paralytic and not of the spastic form, and as due to disease of the cilio-spinal centre in the lower cervical and upper dorsal portion of the cord, or to disease at the origin of the pupil-dilating fibres in the front part of the aqueduct of Sylvius. The myosis is often extreme in degree, or "pin-hole," as it is then called; yet the pupil may react to light and on convergence. Mydriasis, except as part of a third-nerve paralysis, is seldom seen in tabes, but the pupil may be of normal size.

In tabes the pupils are of somewhat different sizes in a considerable number of the cases, both in the early and in the late stages. Berger[1] observed in thirty per cent. of his cases an elliptical shape of the pupil, the long axis running about vertically, as a rule.

A familiar and important pupil-symptom of tabes—although not altogether peculiar to it—is that known by the name of Argyll-Robertson, of Edinburgh, who first directed attention to it.[2] The phenomenon consists in this, that the pupil, although as a rule contracted, does not respond to light by further contraction, or, if so, but slightly, yet does become more contracted on convergence of the visual axes (or on accommodation). Myosis need not always be present with the Argyll-Robertson pupil; the pupil may be of normal size, or even dilated.

A very generally accepted explanation of the Argyll-Robertson pupil refers it to a degeneration of Meynert's fibres, which form that part of the centripetal path for the pupillary reflex which lies between the point in the optic tract where that path leaves the tract and the aqueduct of Sylvius. It is held by some that the disease in these fibres is primarily due to an ependymitis of the wall of the fourth ventricle, in which they run. But, as has been pointed out by Heddaeus[3] and by Bevan Lewis,[4] while this explanation would answer were all instances of this symptom binocular, it cannot be the true one, inasmuch as the symptom is sometimes monocular. Lesion of Meynert's fibres on one side only would not interfere with the direct pupillary reflex, which could then be brought about by means of the centripetal reflex path passing by the opposite optic tract and opposite fibres of Meynert to the sphincter nucleus of the opposite third nerve, and thence to the sphincter nucleus of the affected side by the intranuclear fibres which connect the two third-nerve nuclei. Both of these

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[1] Loco citato, p. 346.

[2] Edinburgh Medical Journal, 1869, xiv. p. 669, and 1870, xv. p. 487.

[3] Archiv für Augenheilkunde, xxvii. S. 88.

[4] British Medical Journal, April 25 and May 2, 1896.

authors therefore hold that the disease causing the Argyll-Robertson pupil is a nuclear one, and this is Jessop's [1] view also. Heddaeus believes that in some cases the disease may be fascicular rather than nuclear. He thinks it probable that there is no direct connection between the nucleus for accommodation and that for the sphincter iridis, but that the ramus iridis of the third nerve is formed of two roots, one springing from the sphincter nucleus and the other from the accommodation nucleus. Destruction, then, of the root springing from the sphincter nucleus, equally with destruction of the nucleus itself, would give rise to loss of the pupil-reflex on the same side. Yet there is in this theory the difficulty that the phenomenon of the Argyll-Robertson pupil is very frequently associated with myosis. In these cases the loss of the pupil-reflex cannot be due to nuclear or to fascicular disease alone, for were this so, by reason of the action of the sympathetic, mydriasis and not myosis would be present. To obviate this difficulty a concomitant condition of irritation of the pupil-fibres in the optic nerve (Heddaeus [2]), vaso-motor action (Guillery [3]), and a tonic innervation of the iris on part of the third nerve (Oebeke [4]) have each been called upon.

It is evident, indeed, that we have still much to learn before a wholly satisfactory explanation of the occurrence of this valuable clinical symptom can be given.

The symptom is one of those most regularly found in tabes. It is very often an early or initial symptom, and it continues through all the stages of the disease. But it is sometimes entirely absent.

The writer is led to think by some cases which have come under his notice, that the full value of the Argyll-Robertson pupil as a very early premonitory symptom of serious general nervous disease (general paralysis or tabes) has not yet been fully appreciated, and that it perhaps plays this *rôle* more frequently than is generally supposed. An interesting question too as regards this symptom is, whether or not it may be present without foreshadowing the approach of any general nervous disease,—*i.e.*, as a purely local symptom. The writer would be inclined to give a negative reply to this question, and in this opinion he is supported by that of Dr. Byrom Bramwell, kindly expressed to him by letter. Dr. Bramwell says, "I should certainly say, in my experience, the Argyll-Robertson symptom is significant of some general nervous affection. I only remember to have once met with the condition unassociated with other symptoms and signs of nervous derangement, and I do not feel definitely sure about that case. In the vast majority of instances in which I have found the Argyll-Robertson pupil present, the case has been one either of locomotor ataxia or of general paralysis of the

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[1] Transactions of the Ophthalmological Society of the United Kingdom, xi. p. 182.

[2] Klinische Monatsblätter für Augenheilkunde, 1893, S. 98.

[3] Ueber topische Diagnostik der Pupillarerscheinungen bei der Tabes Dorsalis, Deutsche medicinische Wochenschrift, 1892, Nr. 52.

[4] Allgemeine Zeitschrift für Psychiatrie und psychisch-gerichtliche Medicin, l., 1894, S. 148.

insane." Gowers, in speaking of nerve-degenerations the result of syphilis,[1] says, "Among the motor palsies, one of the most important to the physician, although the least important to the patient, is the isolated loss of the light-reflex of the iris, with which the name of Argyll-Robertson is connected. I have notes of only twenty-two cases in which this symptom existed apart from other degenerative affections of the nervous system, but I believe that it is much less rare than this number might suggest. It is seldom looked for unless other symptoms suggest degenerative disease. Of these twenty-two cases, syphilis was certain in twelve and highly probable in three others, while two more had a history of a venereal sore. In six cases there was no history or evidence of either syphilis or chancre; nevertheless, two of them had had an attack of sudden hemiplegia in early adult life, without any indication of a cause of embolism,—an event that always affords ground for suspecting syphilis. From these facts we may feel confident that syphilis existed in two-thirds of the cases, and we may suspect that it existed in three-quarters. The relation of this symptom to syphilis makes it one of great practical value." And then Gowers adds the significant statement, "It [the Argyll-Robertson symptom] proves that a degenerative process is at work in the nervous system, and it raises a strong presumption that this process is the result of syphilis. It is true, the dependence on central degeneration has not been proved by observation, but the indirect evidence of it is very strong." Apart from the connection with syphilis pointed out by Gowers, the Argyll-Robertson symptom has been noted by some authors in combination with other symptoms of disease, but where at the time there were no definite indications of locomotor ataxy or of general paralysis. The writer submits that the question which still remains to be answered is: Is not the Argyll-Robertson symptom, whether alone or in combination with other symptoms, always a sign of tabes or of general paralysis, it may be in a very early stage, and possibly sometimes of other less well-defined diseases of the nervous system?[2] This question will not be an easy one to answer, for the opportunity of keeping suitable cases under observation for the necessary length of time is rare.

Neither the Argyll-Robertson pupil nor primary optic atrophy occurs in peripheral neuritis, a disease which is liable to be occasionally mistaken for tabes.

Boedecker relates a case[3] of tabes in which the disease began with double sixth-nerve paralysis, soon followed by Argyll-Robertson pupil, and five years later the first certain signs of tabes presented themselves (loss of knee-jerk, lightning pains, etc.).

The pupillary reflex to sensitive stimuli—the sympathetic reflex—is lost much later than the light-reflex, and finally the reaction on convergence also disappears.

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[1] Lettsomian Lectures on Syphilis and the Nervous System, London, 1892, S. 47

[2] Deutsche Zeitschrift für Nervenheilkunde, 1893, iv.

[3] Archiv für Psychiatrie und Nervenkrankheiten, xxii. 813.

In testing for the light-reflex it is important that the skin of the eyelids or face be not touched, whereby the sympathetic reflex may be released, and, owing to the consequent dilatation of the pupil, a false conclusion may be arrived at. The light-reflex is best tested in the ophthalmoscope room, or other darkened room, by the aid of artificial light which can be alternately directed on and off the eye. A common and much-to-be-deprecated method among medical men is to look for the light-reflex by opening and closing the patient's eye with a finger or thumb applied to the upper lid.

*Paralysis of Accommodation.*—Paralysis of accommodation without paralysis of the sphincter pupillæ is a rare symptom in tabes. Galezowski has reported some such cases[1] in which the pupil was normal, but in which, in one eye usually, with paralysis of accommodation there was loss of sensation of the skin around the orbit, and Berger[2] has seen similar cases. Paralysis of accommodation is more common in the late than in the early stages of the disease.

*Narrowing of the Palpebral Fissure.*—Narrowing of the palpebral opening due to a slight drooping of the upper lids, hardly to be called ptosis, was first pointed out by Jacobson[3] as sometimes occurring in tabes along with the myosis. It is held to be due to paralysis of the sympathetic (sympathetic ptosis), is usually binocular, and the frequency of its occurrence increases as the disease advances.

*Imperfect Closure of the Eyelids, with Twitchings in the Orbicularis Palpebrarum.*—Berger[4] has been the first to call attention to the fact that a few moments after a tabetic patient has closed his eyelids fibrillary twitchings take place in the orbicular muscle. If only one eye be closed, the twitchings will occur in the opposite orbicular muscle as well, and the greater the effort made to close the eyes the more marked will be the twitchings. Such eyelid twitching may sometimes be seen in other nervous disorders, and sometimes in healthy persons. Berger thinks they are, in tabes, the result of a somewhat imperfect power of closing the eyelids, due to very slight facial paralysis, and hence their marked character in that disease.

*Epiphora.*—Epiphora is, according to Berger, not a rare symptom in tabes. He has noted its occurrence in nearly fifty per cent. of his cases and in all stages of the disease. But Gowers and Oppenheim, in their manuals, do not mention the symptom at all, while others[5] expressly state it to be a rare symptom, an opinion which coincides with the writer's experience. Panas[6] once saw epiphora as the first symptom of tabes. It may give rise to injection of the conjunctiva, to marginal blepharitis, and to eversion of the lacrymal punctum. This epiphora is caused partly by difficulty in the

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[1] Société de biologie, February 18, 1888.
[2] Loco citato.
[3] Augenaffectionen bei Allgemeinleiden, S. 64.
[4] Loco citato, p. 392.
[5] Terson, Gazette médicale de Paris, 1894, No. 33.
[6] Gazette médicale de Paris, August 18, 1894.

flowing off of the tears, consequent on debility of the orbicular muscle; but it is probably chiefly due to hypersecretion, in analogy with the hyperidrosis of the face.

*Reduction of the Intra-Ocular Tension.*—This is a symptom to which, as yet, Berger[1] alone has directed attention. Out of one hundred and nine cases of tabes he found hypotony present in thirty-five, and in only twelve of these was there myosis. Berger derives the symptom from paralysis of the sympathetic, relying on the experiments of Adamük, who showed that division of the sympathetic reduced, while stimulation of it elevated, the intra-ocular pressure.

## HEREDITARY ATAXY, OR FRIEDREICH'S DISEASE.

This disease has few eye-symptoms, a fact which is of some diagnostic importance. Ataxic nystagmus is indeed the only one which occurs with any constancy, as was pointed out by Friedreich in his original description of the disease.[2] When the eyes are at rest there is no nystagmus, but when they are fixed on the finger placed a short way in front of them (convergence) slight oscillations of the eyeballs may be observed, and these oscillations are still more marked when the eyes are caused to follow the fixation object from side to side (ocular ataxy).

Optic atrophy can hardly be reckoned among the symptoms of the disease. The incipient stage has been noted by Oliver[3] in one case along with contracted fields, and very occasionally by other authors, but it is evidently of rare occurrence in Friedreich's disease. Paralyses of orbital muscles do not occur, nor does any pupil-symptom.

## MYELITIS.

Acute inflammation of the cord, apart from the inflammation of its meninges (cerebro-spinal meningitis), of which we have already spoken, may be associated with optic neuritis. Instances of this have been recorded by Noyes,[4] Steffan,[5] Sharkey and Lawford (with post-mortem),[6] Dreschfeld (with post-mortem),[7] and others. In Sharkey and Lawford's case, as also in Dreschfeld's, the papillitis preceded the symptoms of spinal inflammation by thirty days, and in a case reported by Achard and Guinon[8] by six days, and here the spinal affection commenced in the lower parts of the cord and ascended. An important paper on this subject is that of Elschnig,[9] who records three

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[1] Gazette médicale de Paris, p. 394.

[2] Archiv für pathologische Anatomie und Physiologie und für klinische Medicin, lxviii., II., 1876.

[3] Keating's Cyclopædia of the Diseases of Children, iv. p. 202.

[4] Archives of Ophthalmology, 1880, p. 199.

[5] Bericht der ophthalmologischen Gesellschaft zu Heidelberg, 1879.

[6] Transactions of the Ophthalmological Society of the United Kingdom, iv. p. 232.

[7] British Medical Journal June 2, 1894.

[8] Archives de médecine expérimentale et d'anatomie pathologique, 1889, No. 5.

[9] Archiv für Augenheilkunde, xxvi. S. 56.

cases in which optic neuritis, associated with intense pain (for which the author offers no explanation) in and about the eyes, especially on passive pressure or on motion of the eyeballs, and with amaurosis, was followed by symptoms of acute myelitis. Of these cases, two underwent satisfactory cures, both as regards eyesight and spinal-cord symptoms, and in the third the recovery was only partial in respect of each of these. (Elschnig in the same paper records a case of optic neuritis associated with encephalitis, an extremely rare diseased condition.) Another valuable paper on the subject is that of Katz,[1] who has had a case with post-mortem. The optic nerve seems usually to become inflamed before the spinal cord, but the myelitis may precede the optic neuritis, or optic nerve and spinal cord may be simultaneously attacked. The relation of the optic neuritis and myelitis to each other, there can be little doubt, is nothing more than that each is a manifestation of the presence in the system of one and the same toxic influence, whatever it may be; and, should the optic nerve succumb to this influence earlier than the spinal cord, we can but conclude, in the present state of our knowledge, that it is because, in the particular case, the optic nerve is a *locus minoris resistentiæ*. The rheumatic diathesis, exposure to cold, epidemic influenza, and syphilis are among the causes assigned in some cases, while in others none could be ascertained. Blindness comes on with a rapidity which is remarkable and alarming, but, after an interval of some ten or fourteen days, vision usually begins to return; and, therefore, serious as is the aspect of these cases, the prognosis is not entirely bad, for a large proportion of them undergo at least partial cure, in respect both of the spinal and of the optic-nerve affection.

Even complete recovery of sight has been observed. But, on the other hand, atrophy of the optic nerve with absolute amaurosis is not an uncommon result.

Gowers[2] points out as noteworthy, that most of the cases of acute myelitis accompanied by optic neuritis have been instances of disseminated myelitis, a form that suggests a cause acting widely on the nervous system.

If the cervical portion of the cord be inflamed, pupillary symptoms—irritation mydriasis or paralytic myosis—are apt to be present.

It was for a long time held that those symptoms of nerve-disease which sometimes slowly develop after railway and some other accidents are due to a myelitis or a meningo-myelitis; but we now recognize that the so-called railway spine is a condition which belongs to the category of functional neuroses, and is better termed traumatic neurosis,—a disorder of the whole central nervous system, but mainly of the brain.

Yet it must be admitted that in a very small proportion of cases these accidents do give rise to hemorrhages in the cord and its meninges, or to myelitis, secondary, probably, to such hemorrhages. Associated with these

[1] Archiv für Ophthalmologie, xlii., 1, S. 202.

[2] Diseases of the Nervous System, 2d ed., i. p. 319.

changes it is said that optic neuritis and optic atrophy sometimes occur; but it is a question whether these ocular affections may not rather be due to direct injury of the nerve at the optic foramen by concussion, or to a basal meningitis. (See also below, under Injuries of the Spinal Cord.)

### SYRINGOMYELIA AND MORVAN'S DISEASE.

Into the vexed question of the identity, or otherwise, of these diseases the writer will not enter. One eye-symptom is common to both of them, and is frequently present,—namely, a concentric contraction of the field of vision without ophthalmoscopic changes. It seems to be not yet quite certain whether the alteration in the field is due, at least sometimes, to attendant hysteria, or is always a symptom of the organic disease, as such.

Déjerine and Tuiland[1] found marked contraction of the field in seven cases of syringomyelia in which hysteria could be definitely excluded. The fields were contracted for all colors, but chiefly for green; while the contraction for white was much less marked. Inequality in the pupils has sometimes been noted. Oppenheim[2] alone states that in his experience nystagmus is by no means rare, and that optic neuritis has sometimes been observed.[3]

### MYOTONIA CONGENITA, OR THOMSEN'S DISEASE.

In some cases of this rare affection, the nature of which is still obscure, the external musculature of the eyes affords symptoms, although the intrinsic muscles are never disordered. The opening and closing of the eyelids may be difficult. They cannot be closed or opened at one stroke. In one case,[4] for example, the patient required sixteen seconds to get his eyelids quite open, and sometimes successive jerky motions are required to effect the closure. Also, when open, the upper lid is apt to be retracted and the eyelids elevated, and, as in Graves's disease, the upper lid does not readily follow the downward motions of the eyeball. The various associated motions of the eyeballs, too, are impeded in some cases. Raymond alone makes any mention[5] of a derangement of vision in connection with Thomsen's disease. In two cases he noted transitory amblyopia, or even amaurosis, following or accompanying spasm of the muscles of the body, and in one case following motions of the head. In one of these cases, too, the author observed hypertrophy of the internal recti and of one external rectus muscle.

### LANDRY'S DISEASE, OR ACUTE ASCENDING PARALYSIS.

The pathology of this disease has not yet been definitely settled. In some cases slight inflammatory changes have been found in the spinal cord;

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[1] La Semaine médicale, 1890, No. 30.
[2] Lehrbuch der Nervenkrankheiten, Berlin, 1894, S. 257.
[3] Loco citato, p. 258.
[4] Friis, Neurologisches Centralblatt, 1892, S. 40.
[5] Gazette médicale de Paris, June 27, 1891.

in others the spinal cord has been healthy, but some parenchymatous changes have been found in the peripheral nerves; while in by far the greater number of cases no diseased changes whatever have been seen in the nervous system. Eye-symptoms are rarely present in Landry's disease, but there may be paralysis of some of the orbital muscles, paralysis of accommodation, dilated pupil, or loss of the light-reflex.

## INJURIES OF THE SPINAL CORD.

That condition which was described by Erichsen as "railway spine," but which has come to be known under the title of traumatic neurosis, and is now regarded as the result, for the most part, of mental shock rather than of organic lesions of the brain or spinal cord, is accompanied very frequently by certain functional eye-symptoms. A description of this state does not come within the scope of this chapter.

In those much rarer cases of organic injury to the cord, or of myelitis, or of hemorrhage in or inflammation of its membranes, following on railway and other accidents, organic eye-disease seldom results. As regards optic neuritis and optic atrophy, which used to be held as frequent consequences of spinal injuries, Thorburn [1] states that slight spinal injuries are very common, but there is no evidence that they tend to be followed by changes in the optic disk. Such injuries are, however, in rare cases followed by chronic meningitis and myelitis, and in the latter condition there is an *a priori* probability or possibility that optic neuritis may supervene. Page, too,[2] says there is no evidence of pathological change in the optic nerves being common in cases of spinal injury and nervous shock. Allbutt [3] found hyperæmia of the optic papilla with some indistinctness of its margins and over-filling of the retinal vessels, but no optic atrophy, nor tendency of the condition to pass into optic atrophy, and referred the changes he found to secondary subacute meningitis at the base of the brain. It is now well-nigh a quarter of a century since Allbutt published these views, and little or no confirmation of them has since appeared; yet even Allbutt did not see optic atrophy as the result of spinal injury.

Inexperienced observers must be careful not to be led astray in the diagnosis of optic atrophy in railway and other cases by the presence of the contracted fields so often found in traumatic neurosis, combined with a somewhat pale, yet in fact quite normal, optic disk. Also they should not rush into the diagnosis "optic hyperæmia," or "optic neuritis," because there are some complaints of vision, with the presence of a high-complexioned yet normal optic papilla.

If the lesion be in the lower cervical region of the cord, the pupils are apt to be contracted from sympathetic paralysis.

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[1] A Contribution to the Surgery of the Spinal Cord, London, 1889, p. 178.

[2] Railway Injuries in their Medico-Legal and Clinical Aspects, London, 1891, p. 43.

[3] On the Use of the Ophthalmoscope, London, 1871, p. 197.

# OCULAR LESIONS DEPENDENT UPON DISORDERS OF THE SECRETORY AND EXCRETORY ORGANS.

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## OCULAR LESIONS IN DISEASES OF THE URINARY SYSTEM.

### I. BRIGHT'S DISEASE.

OCULAR lesions have been described in association with all forms of disease of the kidneys, but occur with much greater frequency in certain well-recognized varieties. The older observers, in the beginning of the nineteenth century and earlier, were cognizant of and wrote concerning the occurrence of defect or loss of sight in patients suffering from dropsy (Wells, 1812), as, for example, that occurring in puerperal women, or that following scarlatina. Bright, in the third decade of this century, published his "Reports of Medical Cases," in which he demonstrated that in many cases of general dropsy the urine contained albumen, and the kidneys after death showed signs of disease. In subsequent investigations of the subject, Bright[1] and his co-workers at Guy's Hospital observed and reported cases of albuminuria in which marked defect of sight occurred. In thirty-seven patients with albuminuria in whom there were uræmic manifestations, loss of sight occurred in four. In some of the cases the onset of amaurosis was one of the earliest as well as the most striking of the clinical features. In the next few years several authors published their observations concerning the occurrence of defects of vision in patients with albuminuria (Osborne, 1837, Addison, 1839, Christison and Gregory, 1839, Rayer, 1840).

Landouzy[2] (1849), although some years later than Bright, had, apparently, without knowledge of Bright's work, made the discovery that loss of sight was occasionally associated with diseased conditions of the kidneys. He did not, however, consider that any causal relation existed between the renal disease and the ocular symptoms. Türck,[3] in 1850,

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[1] Bright, Guy's Hospital Reports, 1836, and subsequent volumes.
[2] Gazette médicale, No. 42.
[3] Zeitschrift der Gesellschaft von Wiener Aerzte, Nr. 4.

was the first to show that the amblyopia of albuminuria was, in some instances, due to visible structural alterations in the retina. He examined microscopically the retina removed post mortem from a case of albuminuria in which one of the clinical symptoms had been loss of vision, and gave the earliest description of the histological changes in albuminuric retinitis. His discovery was soon confirmed by other investigators, and a few years later the opinion was put forth by Virchow that the so-called uræmic amaurosis was attributable to gross disease of the retina, a view which later inquiries proved to be very partially correct.

Heymann and Zenker[1] and Virchow[2] (1856) by their investigations added considerably to the knowledge of the subject. The former did much to establish the importance of retinal disease in albuminuria, while Zenker's and Virchow's labors dealt chiefly with the pathological anatomy of the retina. To Liebreich[3] (1859) belongs the credit of the first detailed description and representation of the ophthalmoscopic appearances of albuminuric retinitis.

Since that time very numerous additions to the literature of the eye affections in Bright's disease have been made. In this, as in other subjects, however, the growth of knowledge has not been altogether commensurate with the increase in the amount of literature. There is still room for investigation, especially in reference to the exact relations of the ocular lesions to the general disease.

## URÆMIC AMAUROSIS.

It has been known for a very long time that in some cases of Bright's disease cerebral symptoms are predominant. Such manifestations of the disease have been termed "uræmic," although the theory as to their causation implied by the name cannot now be wholly accepted (Fagge). Discussion of the various explanations of the condition of uræmia would be out of place in this article.[4] There seems no doubt, however, that it is produced by the poisonous action on the nervous centres of materials accumulated in the blood as the result of defective excretion by the kidneys, but it is still uncertain whether this action is excited by one substance or by more (Fagge). Uræmic amaurosis is undoubtedly one of the cerebral symptoms of Bright's disease, although, as already mentioned, there was at one time a disposition to explain loss of sight in all cases by retinal lesions.

Epileptiform convulsions, coma, and amaurosis are sometimes classed as the acute manifestations of uræmia, the chronic symptoms being headache, giddiness, or slowly-developing stupor, and vomiting. Amaurosis is

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1 Heymann and Zenker, Archiv für Ophthalmologie, Bd. ii. Abth. 2, S. 137.

2 Archiv für pathologische Anatomie und Physiologie, Bd. x. S. 170.

3 Liebreich, Archiv für Ophthalmologie, Bd. v. Abth. 2, S. 265.

4 The different hypotheses are given and critically examined in text-books of medicine such as those by Hilton, Fagge, and Osler, and in monographs on diseases of the kidneys.

one of the most remarkable of the former group. It may be unaccompanied by other serious symptoms, but generally there is, in addition, headache and vomiting. Sometimes the amaurosis is preceded or accompanied by convulsions; the patient may be seized with a fit, and after recovering consciousness is found to be blind. Wagner[1] states that amaurosis may occur shortly before a convulsive attack.

The onset of the amaurosis is usually sudden, and the loss of sight bilateral and complete; even perception of light is abolished. In some instances it is stated that vision fails rapidly, but that a period of several hours elapses before it is wholly lost. Perception of light is sometimes retained throughout the attack. The duration of the blindness varies. Sight is usually regained in twelve to twenty-four hours, but occasionally not for two or three days. Recovery of vision is, as a rule, complete. Recurring attacks of amaurosis have been met with during the course of a case of renal disease, and in such it is said that permanent defect of vision may ensue.

The condition of the pupils in uræmic blindness is a point of interest, and there has been some want of agreement thereupon among writers. Generally the pupillary reaction to light is maintained; the pupils are rather dilated, but sometimes quite normal in size, and even where complete blindness is present they may still contract on exposure to light (Leber, Gowers, Schmidt, and others). In some cases, however, the pupils are widely dilated and motionless to light (von Graefe and others). It does not seem possible, with the evidence at present available, to determine whether, in cases of complete blindness, it is more common to find the pupillary light-reflex retained or abolished. The evidence seems to point to retention of pupillary reaction in the majority of cases. The question has an important bearing on the immediate causation of the blindness. If the pupillary reaction is retained while absolute blindness exists, the toxic effect must be exerted upon some part of the visual tract higher than the corpora quadrigemina. If the pupillary light-reflex is lost, the action of the poison may be upon both central and peripheral parts of the visual tract, or, as has been stated, only upon the peripheral parts (optic tract and nerve). It cannot yet be said that the cortical origin of uræmic amaurosis is fully established.

Ophthalmoscopic examination in uræmic amaurosis in certainly the large majority of instances reveals nothing abnormal—excluding cases with pre-existing retinal changes. A few exceptions to the rule are, however, on record (Gowers,[2] Wilkinson[3]). In a case reported by Litten, in which uræmic amaurosis occurred in a patient already affected by albuminuric retinitis, it is stated that the swelling of the optic papilla, and the adjacent opacity of the retina, became more marked during each of several

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[1] E. Wagner, Handbuch der allgemeine Pathologie, Leipzig.
[2] Gowers, Medical Ophthalmoscopy, 3d ed., London, 1890.
[3] Wilkinson, American Journal of Ophthalmology, 1897, p. 155.

uræmic attacks, and underwent recognizable diminution in the intervals. The association of albuminuric retinitis and uræmic amaurosis is, however, not common.[1]

The prognosis as regards recovery of vision in amaurosis is good. Relief of the uræmic state by active treatment generally leads to a rapid and sometimes sudden return of sight. Prolonged duration of the blindness and recurrent attacks are unfavorable signs prognostically. Permanent defect of sight, with contraction of the field of vision, may ensue, probably caused by atrophic changes in the optic nerves. Further information is wanted concerning the after-history, as regards vision, of patients who have had uræmic amaurosis, and in whom the renal disease has not proved rapidly fatal—*e.g.*, puerperal women.

Uræmic blindness may occur in any of the various forms of renal disease in which there is sufficient interference with excretion by the kidneys to load the blood with those poisonous materials, whatever their exact nature may be, which induce the uræmic condition. It, in common with other uræmic symptoms, is usually, though not invariably, preceded by a notable diminution in the quantity of urine, or by its almost complete suppression. It is a remarkable fact, to which Fagge calls attention, that uræmia is seldom met with in persons advanced in years; perhaps this suggests that a predisposition on the part of young subjects is one factor in its etiology.

Uræmic amaurosis is more common in the acute varieties of kidney disease. That form of renal disease known as tubal or parenchymatous nephritis is caused by, *inter alia*, scarlet fever, small pox, and some other eruptive fevers, pregnancy, especially in primiparæ, and exposure to cold. As far as records of cases tell us, nephritis induced by scarlatina and that occurring during pregnancy seem to be more frequently accompanied by uræmic amaurosis than nephritis due to other causes. Amaurosis does, however, occur in the acute nephritis following exposure to cold, and is also met with in instances of chronic Bright's disease in which an acute attack supervenes. Leber[2] refers to a case reported by Adler in which acute nephritis during small-pox was accompanied by uræmic amaurosis and deafness, followed by recovery of both senses.

### RETINITIS AND NEURO-RETINITIS.

Among the most characteristic and widely-known ocular manifestations of general disease must be classed the retinitis and neuro-retinitis of albuminuria. The different types of retinal disease which are met with, their ophthalmoscopic appearances, and the histological characters of the affected tissues are described in the section on Diseases of the Retina, in Vol. III.

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[1] Bull, Transactions of the American Ophthalmological Society, 1886, noted uræmic amblyopia in thirty-seven out of one hundred and three cases of albuminuric retinitis and neuro-retinitis.

[2] Leber, Graefe-Saemisch, Handbuch der gesammten Augenheilkunde, Bd. v. S. 955.

# PLATE I.

Fig. 1.

Fig. 2.

Albuminuric retinitis. These two drawings represent the ophthalmoscopic appearances of the right eye of Elizabeth B., at an interval of ten months.

July, 1894. Right eye (upright image). There is some obscuration of the optic disk, but no appreciable swelling. Retinal veins turgid, arteries unduly bright and in places hidden by hazy œdematous retina. Scattered over the central region of the fundus are numerous soft-looking patches of whitish exudation in the retina, and a few small linear hemorrhages. The macular region is occupied by a large irregular patch, consisting of whitish exudation, arranged in broken radiating lines and dots, and hemorrhage.

May, 1895. Right eye (upright image). The disk is paler, more yellow, and less obscured. The retinal arteries are very bright, slightly shrunken, and compress the veins which they cross. The retinal exudation has almost wholly disappeared, leaving a slight granular pigmentary disturbance behind. At the macula is a dark irregular patch, surrounded by a lighter areola, and near it a few lustrous points of degeneration in the retina. The improvement in vision between the dates of the two drawings was as marked as the change in the ophthalmoscopic appearances (vide page 650). Patient died in September, 1895.

of this System, and but few references to these points will be made in this article. The microscopic appearances of the diseased retina from a case of albuminuria are depicted in Plates IV. and V. Before proceeding to the consideration of the conditions commonly, but not very accurately, termed albuminuric retinitis and neuro-retinitis, certain less obvious but important changes in the retina will be referred to. These changes are frequently, though not invariably, met with in the subjects of chronic Bright's disease. They affect the retinal vessels, are most noticeable in the arteries, and may easily be overlooked unless careful ophthalmoscopic examination be made. Some years ago Gowers[1] described "a notable diminution in size of the retinal arteries in some cases of chronic renal disease, especially of the granular form," in which the ordinary retinal changes of albuminuria are not present. The veins are unaltered in size, but the arteries may be reduced to half their usual breadth, or even less. This diminution in size of the retinal arteries is coincident with a high-tension pulse. These statements have been confirmed by Marcus Gunn,[2] who has published, in a very valuable paper, the results of his observations of the retinal vessels in cases of chronic albuminuria and other conditions in which arterial degeneration occurs. He gives a detailed description of the ophthalmoscopic appearances of the diseased retinal arteries, and by a series of cases, followed to their termination, has very clearly exemplified the significance of these retinal signs.

The arteries are usually narrowed, but irregularly so; a vessel of apparently normal size may become much reduced in calibre for a portion of its course, and then regain its full breadth, or there may be a local increase in size. The arteries are often unusually tortuous. The central light streak of the arteries is much brighter than in health, and this is often more noticeable on the smaller divisions of the vessels than on the main trunks. This unusually bright streak gives the artery an appearance like that of a polished copper or silver wire, and Gunn uses the term "silver wire arteries." There is also some loss of translucency in the artery, so that an underlying vein is hidden where the artery crosses it, and a still more striking appearance is that produced by obstruction to the blood-current in the vein by the pressure of a sclerosed artery which traverses it. These changes are well depicted in the illustrations in Gunn's paper.[3] These abnormal appearances in the retinal vessels, associated with signs of increased arterial tension, are indicative of wide-spread sclerosis of arteries, such as is so frequently present in the subjects of granular kidney. Alterations in the retinal arteries indicative of sclerosis of their coats are

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[1] British Medical Journal, December 9, 1876, and Medical Ophthalmoscopy, 3d ed., p. 209, in which illustrations are given.

[2] Marcus Gunn, Transactions of the Ophthalmological Society of the United Kingdom, vols. xii and xviii. In the latter volume the paper is illustrated by drawings.

[3] Similar changes are shown in the drawings of albuminuric retinitis in Plates I. and II.

commonly seen as part of albuminuric retinitis. In many cases the thickening of the arterial walls and narrowing of the blood stream is very obvious. The importance of slighter but quite recognizable changes in the arteries, which may be present at an earlier stage of renal disease, scarcely needs to be emphasized.

The alterations in the retina comprised under the headings albuminuric retinitis and neuro-retinitis are not only among the most frequent ophthalmoscopic evidences of constitutional disease, but possess a special importance and interest alike for the ophthalmic surgeon and the physician. In the following paragraphs the term "albuminuric retinitis" will be employed in an inclusive sense, and held to embrace all the varieties of retinitis and neuro-retinitis occurring in renal disease.

Retinitis is essentially a complication of the chronic forms of kidney mischief, developing in cases which are primarily chronic, or in those in which an acute nephritis has passed into a chronic stage. Most authorities agree in stating that retinitis is met with only in renal disease of long standing, but some writers mention its occurrence in some of the acute forms of nephritis, notably that accompanying scarlatina. In such instances it is probable that the structural changes in the kidneys are far advanced, although of comparatively short duration.

Retinitis is undoubtedly a more frequent complication of chronic interstitial nephritis (contracted granular kidney) than of any other variety of renal disease, but is not uncommonly associated with chronic tubal nephritis (large white kidney). It is occasionally, though rarely, found in lardaceous disease, and probably only when this has been of long duration, and has given place to well marked atrophic changes in the kidney.

The ophthalmoscopic appearances of the retinal lesions in the different forms of renal disease are not distinctive.[1] Gowers thinks that papillitis, as distinguished from the more usual retinal changes, is more common in cases in which there have been decided symptoms of cerebral disturbance, such as intense headache or delirium. In the patient from whom the drawing Fig. 2, Plate III. was made, intense headache was one of the clinical symptoms. Bull[2] reported two cases in which there was diffuse whitish infiltration of the retinæ in patients suffering from waxy disease of the kidney. He thought that lardaceous degeneration of the retina might account for the peculiar ophthalmoscopic appearances.

The characters of the urine, and other evidences, may enable the observer to diagnose correctly the kind of renal disease present, but in not a few instances this can only be determined post mortem. In the cirrhotic or granular kidney the urine is (generally) increased in amount; it is faintly

[1] It is perhaps not superfluous to remark that retinal changes of whatever kind are not in themselves conclusive evidence of the existence of Bright's disease. Numerous cases have been reported of the occurrence of retinal changes indistinguishable from those of albuminuric retinitis, in other pathological conditions,—*e g.*, intra-cranial tumor.

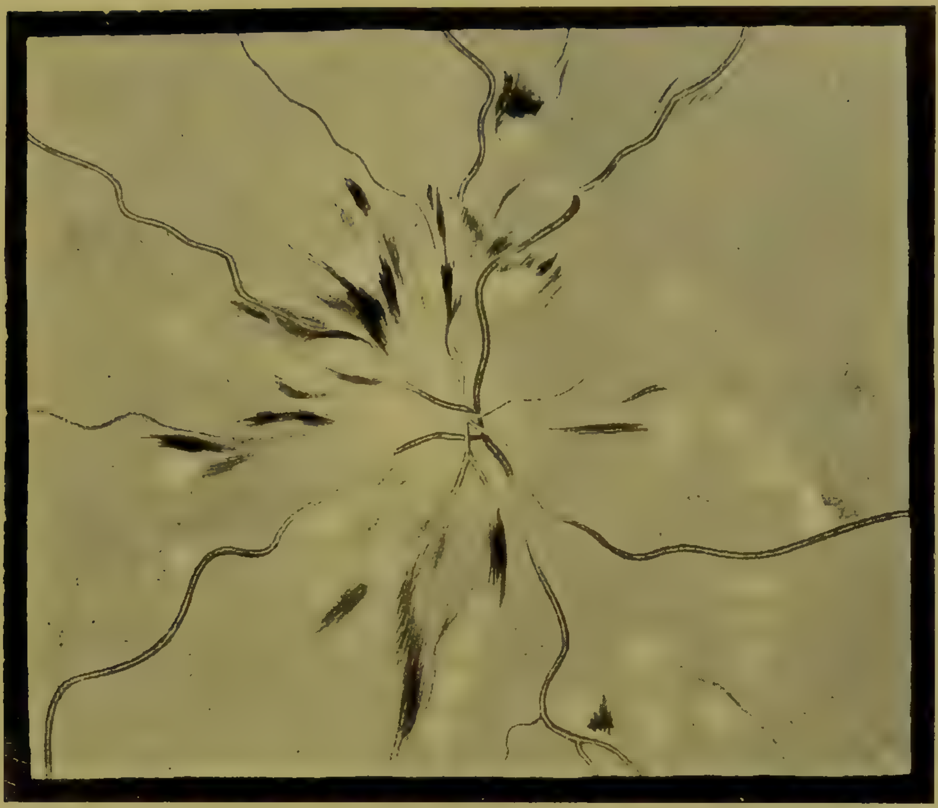
[2] C. S. Bull, American Journal of the Medical Sciences, October, 1879.

PLATE II.

Albuminuric retinitis. (Plate XXXVII., Fig. 84, "The Fundus Oculi, etc.," by W. Adams Frost.) Ophthalmoscopic drawing of the right eye of a man aged fifty-eight, suffering from chronic renal disease. Death from uræmia occurred thirteen months later. The plate shows very characteristically the stellate figure at the macula, composed of radiating broken lines of exudation in the retina. In addition there are three large soft patches of exudation and several hemorrhages in the retina. Note should also be made of the œdematous blurred appearance of the optic papilla, and the compression of the inferior macular vein where crossed by its companion artery. $V = \frac{6}{18}$.

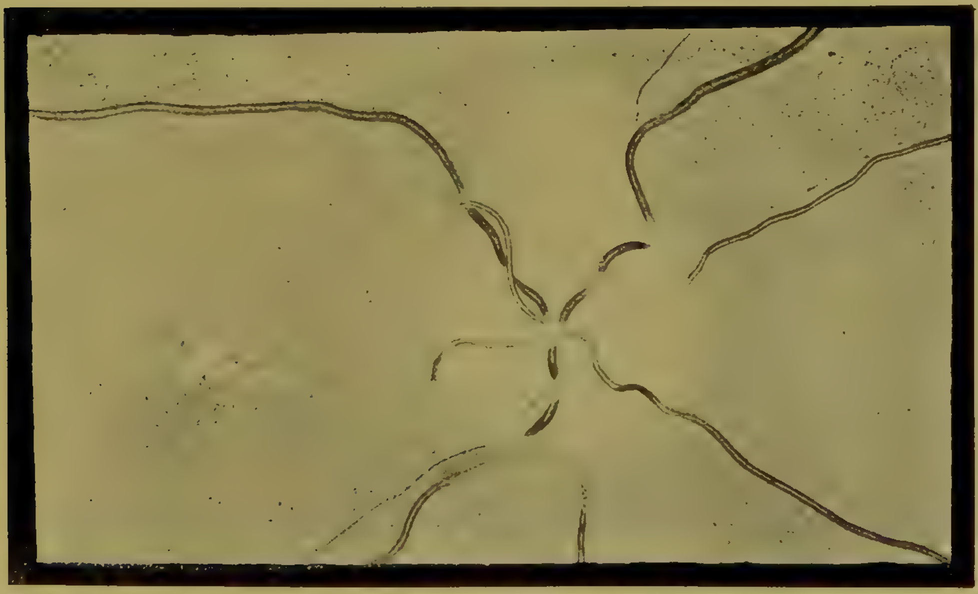
## PLATE III.

Fig. 1.



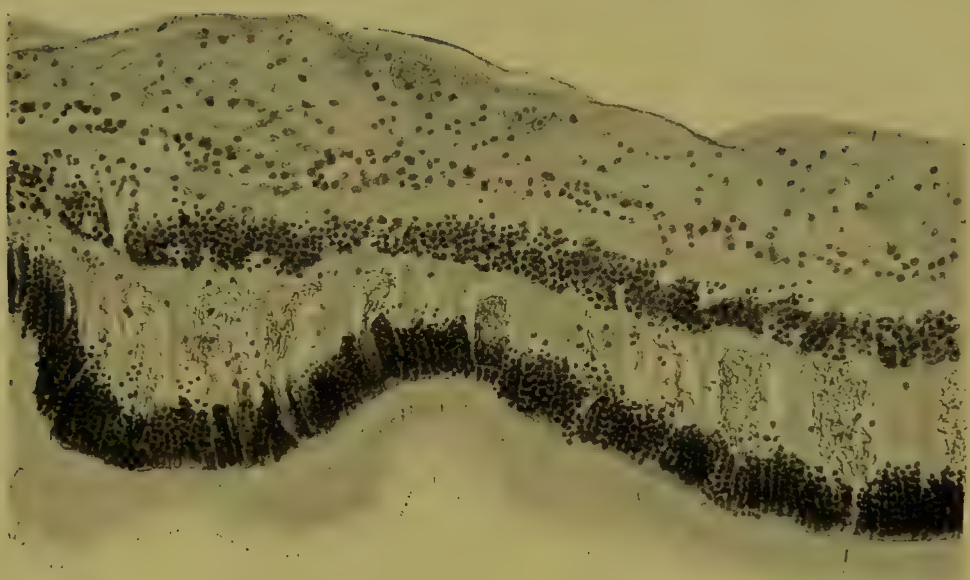
Albuminuric retinitis. (Plate X., Fig. 1, "A Manual and Atlas of Medical Ophthalmoscopy," by W. R. Gowers, M.D., F.R.S.) From a man aged twenty-one, suffering from chronic renal disease, consecutive to an acute attack twelve years previously. Optic disk veiled by pale opacity which extends into the adjacent retina. Many soft white areas and striated hemorrhages in the retina. Veins slightly enlarged, and they and the arteries much obscured at and beyond the margins of the disk. $V = \frac{1}{12}$. The microscopical appearances of the retina in this case are also depicted by Gowers.

Fig. 2.



Albuminuric neuro-retinitis. (Plate IX., Fig. 3, "Medical Ophthalmoscopy," Gowers.) From a case of chronic renal disease (granular kidney). Death from uræmia. The optic papilla is concealed by grayish-red, stippled, and striated swelling. The retinal veins are obscured on the disk, visible as they curve over the swelling, and are again hidden at its edge. The arteries are obscured, and after leaving the papilla are much reduced in size. A few minute white dots are visible in the retina between the disk and the macula, and close to the latter some radiating dots and lines. V = 6 Jaeger.

PLATE IV



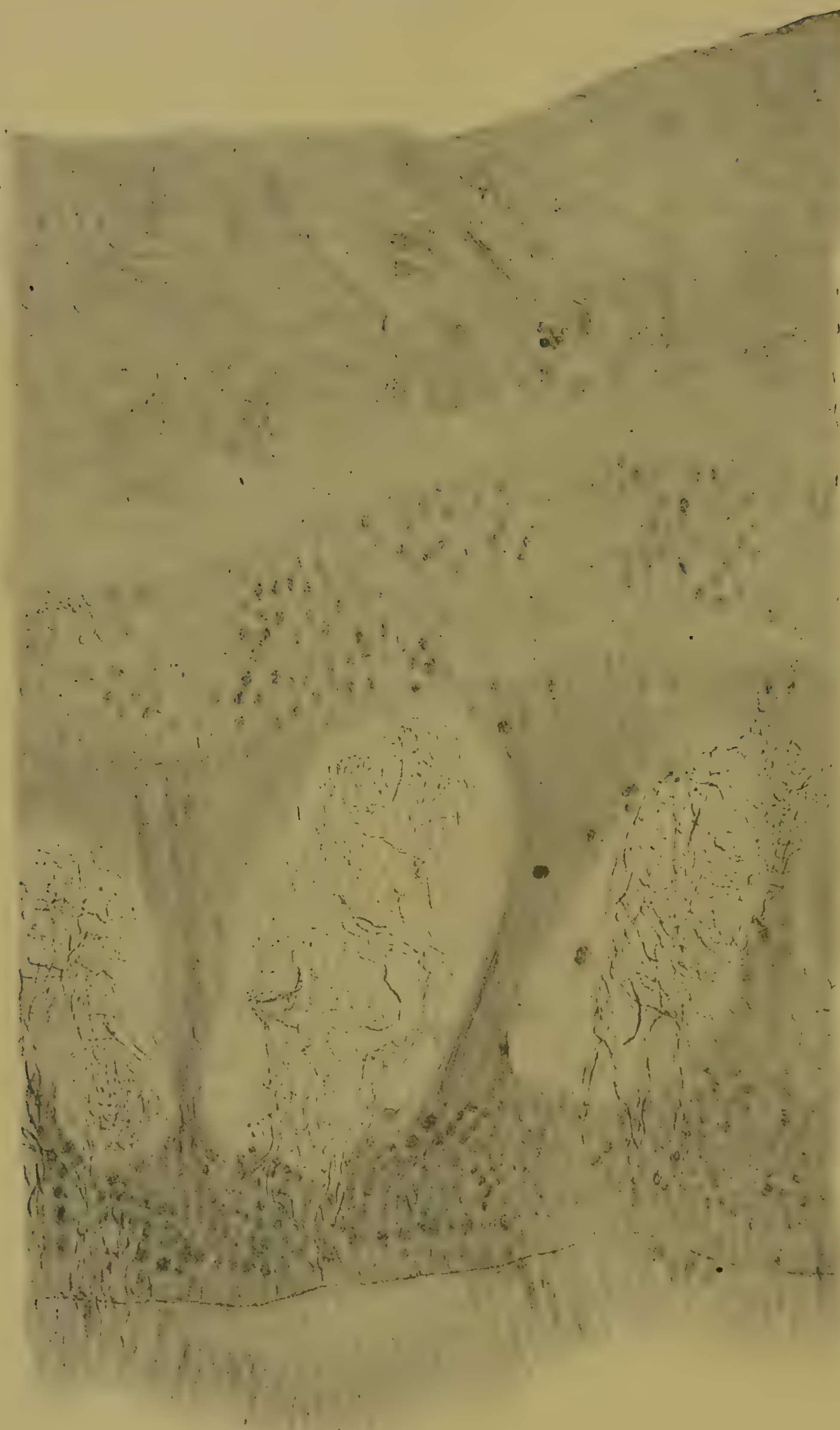
Albuminuric retinitis. × 100. Section of the retina near the macula to show (especially) the swelling of the intergranule layer and the masses of exudation which have pushed aside and distorted the Müllerian fibres.

The drawings (Plates IV. and V.) are from sections of the retina of a man, Francis S——, aged twenty-eight, who died in St. Thomas's Hospital, of renal disease, in January, 1891. He was admitted in a moribund condition, with almost universal œdema; urine scanty and highly albuminous; death three days later.

An imperfect ophthalmoscopic examination, made by the writer a few hours before the man died, revealed "very gross retinal disease in each eye. Numerous white patches, large and small, and some hemorrhage in the macular and adjoining parts of the retina. Optic disks not much swollen."

The report of the post-mortem examination (by Dr. Sharkey) describes the kidneys as "very large and very soft; capsule thin, peeling off easily, leaving a perfectly smooth surface, whitish-yellow in appearance and mottled with distended vessels and small hemorrhages; cortex moderately swollen and of uniform pale yellow aspect, medullary part red."

PLATE V.



Albuminuric retinitis. × 300. Section of the same retina under a higher power. The spaces in the intergranule layer are seen to be occupied by coagulated exudation masses which show a definite fine reticulum. Entangled in the meshes of this are scattered cells, not well shown in the drawing. A mound-shaped collection of cells (inflammatory) is seen separating the bacillary layer from the external limiting membrane at one part. The disproportionate thickness of the intergranule layer is well shown. No hemorrhages are visible in this section.

acid, pale, and clear, with a specific gravity of 1004 to 1010. The quantity of albumen is small, often less than one-half per cent., and its presence may be overlooked unless the examination is made with care. In slight cases, and in the early stage of the disease, albumen may be absent for several days consecutively, and it is noteworthy that it is frequently present in the urine passed after food, while it is absent from the urine passed after a night's rest. The latter is generally submitted for examination. No casts may be discoverable, but if present are usually hyaline in character. In the chronic form of tubal nephritis (large white kidney) the urine is usually abundant, pale, and with a specific gravity varying from 1005 to 1010. It usually contains a large quantity of albumen, although, as the case becomes more chronic, this lessens in amount. Hyaline and granular casts are almost invariably present in large numbers. In lardaceous disease the characters of the urine are very variable. That this must be so will be obvious if it is borne in mind that very frequently lardaceous degeneration is associated with, or follows, other forms of renal disease. The urine is generally excessive, and has a specific gravity much below normal, although varying with the quantity passed. It is pale and clear, with little or no deposit. Albumen is almost always present, and although the amount fluctuates, it is generally large. A few hyaline casts may be found.

The period of the renal disease at which retinal changes develop cannot easily be determined. It is now fairly well established that the retinal lesions never appear prior to evidence of disease of the kidney, as was formerly stated. It is true that retinal changes may be met with in patients with granular kidneys but without albuminuria at the time of onset of the retinitis. Such instances are, however, uncommon, and are cases in which the albumen is present intermittently, or does not appear till a very late stage of the kidney-disease.[1]

In many cases the development of retinitis corresponds in time with the signs of cardiac hypertrophy. Indeed, the frequency of this association led Traube to consider the hypertrophy of the heart as the chief cause of the retinal disease. The association, however, is not a constant one, and the relation between the two conditions is best explained by the fact that both changes are the result of a prolonged period of renal disease. In an analysis of one hundred and three cases of retinitis in chronic Bright's disease, Bull[2] found that in about half that number there was some evidence as to the duration of the kidney-disease, and of these the shortest period was six months.

Albuminuric retinitis is almost always symmetrical, although the severity of the lesions and the time of their onset in the two eyes may vary. Leber[3] states that marked inequality in the retinal changes is exceptional.

[1] Some evidence in favor of the onset of retinitis prior to renal symptoms will be found in a paper by Gand, De la retinite brightique sans albuminurie (Thèse de Paris, 1887), and in another by Eyre (Ophthalmic Review, September, 1897).

[2] Bull, Transactions of the American Ophthalmological Society, 1886.

[3] Leber, Graefe-Saemisch, Handbuch der gesammten Augenheilkunde, Bd. v. S. 584.

Cases of unilateral albuminuric retinitis have been recorded, and although in some of them the second eye probably became affected later, a few instances are known in which the disease attacked one eye only (Cheatham, Yvert, Webster). In Bull's one hundred and three cases, referred to above, retinitis was present in both eyes at the time of examination in fifty-four patients, and in ninety-three of the total number the ocular disease was bilateral before the patients passed from the author's observation.

The retinal complication may occur at any age; in fact, the age of the patient seems to bear but little relation to the development of the retinitis. Chronic interstitial nephritis being much more common in middle-aged and elderly people, a large majority of the cases of albuminuric retinitis are met with in patients over forty years of age. A considerable number of cases in children and adolescents has been recorded, however, and the condition is evidently not so rare among the young as some writers lead us to suppose. Bull mentions that of his one hundred and three cases the youngest was five years old. An instance in a girl aged nine, another in a girl aged twelve, and one in a boy of ten, are recorded by Anderson,[1] Lawson,[2] and Spicer.[3] The illustrations in Plate I. are from a female patient, aged nineteen, under the writer's care.

Although the retinal affection never, or at all events very rarely, occurs until there is advanced renal disease, the failure of vision resulting from retinitis is not infrequently the first symptom for which the patient seeks advice, and hence it is that a good many cases of albuminuric retinitis come first under the observation of the ophthalmic surgeon. The case illustrated in Plate I. was an instance of this, the patient coming to hospital only because of failing vision, although on inquiry it was found that she had suffered from headache and occasional morning-vomiting for some weeks. Another case of very gross retinal disease in a man aged twenty-one, suffering from chronic Bright's disease, was recently under the care of the writer. This patient came because he could not see to do his work as a gardener, and beyond stating that he had suffered from attacks of vomiting six weeks previously, made no complaint of his general health. He died three months later from the kidney affection.

It is confessedly difficult to ascertain the proportion of cases of renal disease in which retinitis occurs, and statistics bearing on this question vary very considerably, the percentages given ranging from about nine per cent. (Wagner[4]) to thirty-three per cent. (Galezowski[5]). Eales[6] examined one hundred hospital cases of chronic Bright's disease, and found retinal

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[1] James Anderson, Transactions of the Ophthalmological Society of the United Kingdom, vol. viii. p. 141.

[2] Arnold Lawson, ibid., vol. xviii. p. 140.

[3] Holmes Spicer, ibid., vol. xviii. p. 145.

[4] Wagner, Archiv für pathologische Anatomie und Physiologie, Bd. xii. S. 218.

[5] Traité des Maladies des Yeux, 1875.

[6] Eales, Birmingham Medical Review, January, 1880.

disease in twenty-eight. In ninety unselected cases examined by Wadsworth,[1] there were twelve (13.3 per cent.) with retinitis.[2] Probably the figures given by Eales and Galezowski represent fairly well the proportion of cases in which retinal manifestations occur, although it is to be borne in mind that all the patients examined by Eales were able to walk about and attend as out-patients at the hospital. The want of agreement in published statistics may be partly explained by the variation in the date of onset of the retinal changes.

The course of the retinal disease follows no definite rule. In the majority of instances the changes persist, although undergoing some alteration in character, to the end. But in not a few cases the retinal lesions retrogress, and sometimes almost entirely disappear. Such improvement occurs in cases in which amelioration of the general condition is effected by treatment, and is most often seen in the albuminuria of pregnancy. In some of the puerperal cases almost perfect vision and a practically normal appearance of the fundus oculi may result. (See notes of case of Mrs. Y. on pp. 655, 656.) The diminution in the retinal disease, especially in cases in which it is recent, is usually but not invariably accompanied by improvement in vision. This is well exemplified in the case from which the drawings in Plate I. were made. Here the vision at the date of the first drawing was 16 Jaeger. Ten months later, when the second drawing was made, it had gone up to 1 Jaeger. Gowers[3] states that the greatest improvement in vision is obtained in cases of the neuritic type of slight degree. In cases of the purely retinal type the severity of the disease frequently damages the retina to such an extent that, even if the exudation masses and hemorrhage undergo absorption, little or no improvement of vision occurs.

Detachment of the retina is one of the more unusual ocular lesions of Bright's disease, and would appear, from available records, to be decidedly rare. Its exact relation to albuminuric retinitis is a point of interest, and one upon which more information is wanted. Is detachment of the retina in these cases a complication of the renal disorder, or is it solely a result of local disease? In all the published cases, so far as the writer is aware,[4] detachment of the retina has occurred in eyes already affected by retinitis, and in those in which the eyeballs were examined post mortem there were obvious changes in the retina. In two of the recorded instances the detachment developed shortly before death from uræmia, and in another it

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[1] Wadsworth, Boston City Hospital Reports.

[2] References to other statistics will be found in Graefe-Saemisch, Handbuch der gesammten Augenheilkunde, Bd. v. S. 585.

[3] Medical Ophthalmoscopy, 3d ed.

[4] Berger refers to a statement of Evetsky, that detachment of the retina occurs in albuminuria independently of retinitis (Les maladies des yeux dans leur rapport avec la pathologie générale, 1892). The writer has failed to find the original statement, after a careful search.

was discovered (its duration being uncertain) nine days before death. These cases would seem to indicate that the onset of the detachment may presage an early fatal termination of the renal disease. The two cases referred to, reported by A. D. Davidson[1] and James Anderson,[2] occurred in children aged fourteen and nine years, and at the post-mortem examination the kidneys were found in an advanced condition of fibroid degeneration. In a case in a woman aged fifty-six the post-mortem revealed the large white kidney.[3] Another case in a child of eleven has been recorded by Dickinson.[4] In this patient the kidneys post mortem presented the characteristic appearances of granular degeneration. Leber[5] and Hirschberg[6] have each recorded a case of detachment of the retina in Bright's disease. Hirschberg's patient was a male aged twenty-two, with severe albuminuria and typical retinitis. Bilateral detachment occurred, and the patient became quite blind before death. West[7] had two cases in young adults of retinitis and detachment; post mortem, the kidneys showed advanced granular degeneration. Wadsworth[8] has published notes of a remarkable case of albuminuric retinitis of pregnancy, in which there was detachment of the retina in both eyes. After the induction of premature labor, and consequent great amelioration of the renal symptoms, the detached retinæ became replaced. It is noteworthy that in this patient there were ophthalmoscopic signs of extensive choroidal changes.

It seems probable that detachment of the retina in Bright's disease is less uncommon than published records indicate. Its occurrence in a late stage of the malady, when the patient is very ill, and in eyes the sight of which is already seriously affected, may well account for its non discovery in a good many instances.

*Prognosis.*—The value in prognosis of renal retinitis is deserving of very careful consideration. Excluding the albuminuria of pregnancy, the appearance of retinal lesions in cases of kidney-disease must undoubtedly be considered a very grave indication. The prognosis as regards vision is bad, but, as has been mentioned, considerable improvement in sight may occur under suitable treatment. It is, however, in reference to the prognosis regarding life that retinal disease is of chief importance, and there is ample evidence to show that the onset of retinal changes in Bright's disease is, in a large majority of instances, an indication of the early termination of life. This is not surprising if it is borne in mind that retinitis

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[1] Davidson, Transactions of the Ophthalmological Society of the United Kingdom, vol. i. p. 57.

[2] Anderson, ibid., vol. viii. p. 141.

[3] Quinlan, ibid., vol. i. p. 58.

[4] Dickinson, Diseases of the Kidney and Urinary Derangements. London, 1877, p. 546.

[5] Leber, Graefe-Saemisch, Handbuch der gesammten Augenheilkunde, Bd. v. S. 576.

[6] Hirschberg, Centralblatt für praktische Augenheilkunde, August, 1884.

[7] West, Transactions of the Clinical Society of London, January, 1895.

[8] Wadsworth, Transactions of the American Ophthalmological Society, 1887, p. 574.

almost always occurs when the renal disease has reached a stage from which recovery is impossible, and hence, even under the most advantageous circumstances, no great prolongation of life can be expected.

Numerous observations and statistics bearing on this question have been published, and a study of them would seem to justify the following statements: (1) that the onset of retinitis in Bright's disease is a certain indication of a fatal termination of the malady, but that (2) the duration of life after the appearance of the retinitis will vary within fairly wide limits, this variation depending partly on the nature and severity of the renal affection and largely upon the conditions under which the patient lives. The statistics upon which the prognosis as to life must be based show that, taking all classes of cases together,—exclusive of pregnancy cases,—a very large proportion, from seventy to eighty per cent., die within twelve months from the date of onset of the retinal disease, and that over ninety per cent. die within two years. The duration of life, as might *a priori* be expected, is greater among patients in good social position, who live under healthful conditions, and who are in a position to carry out dietetic and other forms of treatment, than among the poor who attend hospitals; and it is to be borne in mind that much of our statistical evidence is obtained from the latter class. Bull,[1] Miley,[2] Possaner,[3] Trousseau,[4] Zimmermann,[5] and others have published valuable papers containing statistics as to the duration of life after the onset of albuminuric retinitis, and those specially interested in this subject will find much useful information in the articles by these writers.

The prognosis in cases of retinitis complicating the albuminuria of pregnancy is far less gloomy than in other varieties. This is true both in relation to vision and to life. As has been already mentioned, restoration of useful sight and almost complete disappearance of retinal lesions is not very uncommon in these cases, and permanent recovery from the renal disease is known to occur not infrequently. Several instances of restoration of good vision have come under the writer's notice, and the following case may be quoted: Mrs. Y., aged twenty-nine, had severe albuminuria during her first pregnancy. Vision was very defective, and she was said to be "blind for some weeks." Gradual recovery of vision occurred after the birth of a still-born child, and her health steadily improved. Three and a half years later she was in good health. Vision in the right eye was 6/5 and 1 Jaeger, but with the left she could only read 16 Jaeger in the temporal part of the field, and could not see 6/60. With the exception of a few small whitish dots in the retina on the nasal side of the disk, the right fundus oculi pre-

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[1] C. S. Bull, loc. cit.

[2] Miley, Transactions of the Ophthalmological Society of the United Kingdom, vol. viii. p. 132.

[3] Possaner, Deutschmann, Beiträge zur Augenheilkunde, 1894, S. 22.

[4] Trousseau, Journal des Practiciens, 1895, No. 19.

[5] Zimmermann, Medical and Surgical Reporter, June, 1897.

sented nothing abnormal. In the left eye the disk was pale, atrophic, and hazy, and in the macular region there were degenerative changes in the retina.

Culbertson,[1] from some collected statistics, gives as the result of the albuminuric retinitis of pregnancy: recovery of full vision in (*circa*) sixteen per cent., recovery of partial vision in fifty-eight per cent., blindness in twenty-five per cent. Silex[2] reports twenty-six cases which he had been able to follow; of these, eleven recovered vision above one-sixth; ten recovered vision below one-sixth; five were almost blind.

An important question which arises in cases of albuminuric retinitis in pregnant women is whether or no premature labor should be induced. In some instances there is ample reason for expediting delivery without reference to the involvement of the retina. In others, the onset of severe retinitis may materially assist in the decision. In several of the published cases in which the retinal disease was severe, the induction of labor has been followed by very marked improvement in the ocular condition. Silex,[3] Snell,[4] Alt,[5] Culbertson,[6] and others have published papers upon this subject, and the first named has, from his experience, advised that in nearly all cases in which albuminuric retinitis develops during pregnancy, premature labor should be induced; Snell counsels the artificial termination of pregnancy when the retinal disease is severe.

### DISEASE OF THE UVEAL TRACT IN ALBUMINURIA.

*Choroiditis.*—That the choroid coat may be affected in cases of albuminuric retinitis, producing changes which are visible on ophthalmoscopic examination, is well known; such changes were pictured many years ago by Liebreich in his ophthalmoscopic atlas. Although these signs are not always discoverable on ophthalmoscopic examination, possibly because often obscured by retinal œdema and opacity, it is probable that in a large proportion of cases of retinitis in albuminuria, inflammatory and degenerative processes extend to the choroid, even if they do not arise there at the same time as in the retina. The records of post-mortem examination would seem to confirm this assumption. Apart from this form of disease, a primary albuminuric choroiditis has been described which arises independently of any retinal lesions. Galezowski[7] has observed a form of choroidal disease which is said to resemble, ophthalmoscopically, retinitis pigmentosa, and Magnus[8] in his monograph devotes a chapter to choroidal changes in albu-

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[1] Culbertson, American Journal of Ophthalmology, 1894, p 133.

[2] Silex, Annales d'Oculistique, February, 1895, p. 127; and Münchener medicinische Wochenschrift, 1895, S. 106.

[3] Silex, loc. cit.

[4] Snell, British Medical Journal, June 22, 1895.

[5] Alt, American Journal of Ophthalmology, 1894, p. 141.

[6] Culbertson, loc. cit.

[7] Galezowski, Du Diagnostic des Maladies des Yeux, 1868.

[8] Magnus, Die Albuminurie in Ihren ophthalmologischen Erschein, 1873.

minuria, and describes a case in which, however, there was coincident retinal disease.

*Iritis.*—Iritis and iridocyclitis have been noted as complications of chronic renal disease. The cases are, however, too few to establish a causal connection between the albuminuria and the iritis. Leber[1] has called attention to the association of renal disease and iritis, and has given notes of a case of iritis in a man aged twenty-one, suffering from severe albuminuria. In this patient there was also a disseminated choroiditis.

Evetsky,[2] in the examination of ninety-seven cases of nephritis, found evidence of previous iritis (posterior synechiæ) in 4.1 per cent.

### CATARACT IN CHRONIC RENAL DISEASE.

The coincidence of cataract and albuminuria in elderly people is not uncommon, and the hypothesis has been advanced that renal disease is a cause of cataract. Deutschmann[3] has been the chief supporter of this view, and in a paper on "Nephritic Cataract" published the results of his examination of a large number of cases of cataract in reference to the presence of albumen and casts in the urine. His views have not been generally accepted, however, and statistics by other observers (Landesberg,[4] Rothziegel,[5] Evetsky[6]) have not given similar results. It is not at present established that senile cataract is noticeably more frequent in patients with Bright's disease than it is in those free from kidney mischief. Cataract in young patients with nephritis is so rare that the coincidence can only be considered accidental.

### PARALYSIS OF OCULAR MUSCLES.

A few cases have been recorded in which ocular paralysis has occurred in the subjects of chronic disease of the kidneys. Knies[7] says that such complications of nephritis are by no means uncommon, but he stands almost alone in this statement. He cites three cases which he had seen at brief intervals, and refers to one recorded by Finlayson.[8] The paralysis may affect any one of the nerves supplying the extra-ocular muscles, and although usually passing off quickly, is not unlikely to recur. The cause is probably hemorrhage from degenerate vessels into the nerve-roots or nuclei. Knies's cases were, first, abducens paralysis in a man with albuminuria of fifteen years' duration; two relapses of the paralysis occurred in the few months preceding death; second, left trochlear paralysis in a

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[1] Leber, Archiv für Ophthalmologie, Bd. xxxi. Abth. 4, S. 183.
[2] Evetsky, Archives d'Ophtalmologie, 1887, p. 308.
[3] Deutschmann, Archiv für Ophthalmologie, xxv. 4; xxvii. 1; und xxix. 3.
[4] Landesberg, Archiv für Ophthalmologie, xxx. 4, S. 143.
[5] Rothziegel, Wiener allgemeine medicinische Zeitung, 1886, Nr. 30.
[6] Evetsky, Archives d'Ophtalmologie, 1887, p. 308.
[7] Knies, Die Beziehungen des Sehorgans und seiner Erkrankungen, etc., 1893.
[8] Finlayson, Glasgow Medical Journal, 1877.

patient with contracted granular kidneys, who died six months later; third, a complicated ophthalmoplegia externa, in a male aged twenty-four, with albuminuria of two years' duration. Knies states that the ocular paralyses are indicative of an early fatal termination of the disease, and are suggestive of degeneration of cerebral vessels similar to that found in the vessels of the retina.

### CYCLICAL ALBUMINURIA.

It is now well established that albumen may occasionally be found in the urine of a certain proportion of apparently healthy individuals. This condition has been variously described as functional, cyclical, or physiological albuminuria. Ocular lesions accompanying this malady have been reported, but the records are so scanty that it is evident that disease of any part of the eye sufficiently severe to attract attention must be a very rare complication of what is undoubtedly an uncommon disease.

Two observations concerning retinal changes in cyclical albuminuria have come under the writer's notice. Eales[1] examined fourteen males between the ages of eleven and twenty-eight, suffering from what was believed to be temporary functional albuminuria, and found retinal changes in five,—that is, in rather more than thirty-three per cent. These changes he described as similar in character to those met with in cases of chronic nephritis, and consisting of white degenerative spots and patches. No mention is made of hemorrhage in the retina. Ostwalt[2] has reported in considerable detail two cases of cyclical albuminuria in which ocular lesions were present; one in a female thirty-two years of age, the other in a male aged sixteen. In the former, recurrent hemorrhages from the retinal vessels of the right eye occurred, followed by the formation of a thin vascular connective tissue, attributed to organization of blood-clot, in one part of the fundus oculi, and of limited detachment of the retina in the same region. In the latter, a localized choroido-retinitis of the left eye was found, with recent inflammatory exudation. Twelve days later paralysis of the left facial nerve occurred. Recovery from the retinal and paralytic lesions ensued.

These observations are of interest and importance, although the direct connection of the ocular disease with the abnormal condition of the urine may be open to question. They will at least serve to draw attention to the necessity for careful examination of the eyes in all cases of this form of albuminuria.

## II. DIABETES MELLITUS; SACCHARINE DIABETES; GLYCOSURIA.

This is commonly termed "diabetes," without qualification. The definition given by Fagge[3] is "permanent glycosuria, with polyuria, emaciation, and thirst." The same author writes, "It is not a disease of the

[1] Eales, Birmingham Medical Review, January, 1880, p. 46.

[2] Ostwalt, Wiener klinische Rundschau, 1897, Nr. 41.

[3] Text-Book of the Principles and Practice of Medicine, vol. ii. 1891.

kidneys, of the urine, or of the blood, but is a derangement of the chemical labor of nutrition." Although the urine is abnormal in quantity and constitution, the renal tissue after death is found to be healthy. The urine in this disease has, generally, the following characters: it contains an excessive amount of sugar which, chemically, is indistinguishable from grape-sugar or glycose; the amount of urine is increased, in some cases enormously; instead of two to three pints in twenty-four hours, ten to twenty pints may be passed. Pavy reports a case in which thirty-two pints were measured in one day, and even larger quantities have been recorded. It is much paler than healthy urine, at times nearly colorless, but frequently becomes opalescent on exposure. It has a peculiar odor, compared to the smell of apples. The specific gravity is high,—1030 to 1045.

There are undoubtedly varieties of glycosuria, and the presence of glycose in abnormal quantity, and some increase in the specific gravity of the urine, are probably the only features which are common to all cases.

The ocular lesions occurring in saccharine diabetes, as described by different writers, are very varied. Disease of almost every part of the eye and its surroundings has been noted, and although in some of the recorded cases the ocular lesion can only be regarded as an accidental complication of the general disease, it has been well established that, in regard to certain ocular symptoms, the diabetic condition bears a definite causal relation.

An historical study of this subject is interesting, and, in many respects, instructive, but cannot be dealt with at length in this article. Perusal of a careful and exhaustive paper by Leber[1] will supply those who are interested with an excellent *résumé* of the literature of the subject up to the year 1875, when his communication was published. It will suffice to state here that defect of vision as a complication of diabetes was first definitely recognized[2] in the latter part of the eighteenth century, and that early in the present century such symptoms attracted more attention, and a careful study of them ensued. After the introduction of the ophthalmoscope (1851) knowledge of this, as of so much else in ophthalmology, made rapid strides.

A study of the literature of the subject leaves no doubt that disturbances of vision are of frequent occurrence in diabetes, but we have little to guide us as to their relative frequency. No very satisfactory statistics are available, although several writers have published the proportion of cases met with in their own practices, and others have tabulated numbers of cases from different sources (von Graefe, Hirschberg, Koenig, Lagrange, and others).

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[1] Th. Leber, Ueber die Erkrankungen des Auges bei Diabetes Mellitus. Archiv für Ophthalmologie, Bd. xxi. 3, S. 206.

[2] Leber refers to a case reported in 1688 by Blankaart. A girl affected by diabetes became blind shortly before death. At the post-mortem examination a large intracranial cyst was found which pressed upon the optic nerves.

Very diverse statements are made by authors as to the rarity or frequency of the various ocular lesions; so diverse, indeed, that they must lead readers to the conclusion that the statistics of any one writer cannot be accepted as conclusive except in relation to the particular group of cases that has come under his notice. Thus, one recent writer states that cataract is the most common eye affection in diabetes. Another shows by figures that of cases under his observation paralysis of accommodation was twice as frequent as cataract.

The relation existing between the development of ocular lesions and the severity of the diabetic attack is one concerning which it appears impossible to make any very decided statement. The evidence seems, on the whole, to indicate that, with the possible exception of cataract, the ocular complications are more frequent in cases of a chronic character, in which the general symptoms (emaciation, thirst, etc.) are not very marked. In not a few such instances, indeed, the patient first comes under observation in consequence of the eye-disease.

The ocular complications of diabetes to be considered will be taken in the following order:

1. Paralysis and paresis of accommodation; pupillary anomalies; alteration in the static refraction of the eye.
2. Affections of the extra-ocular muscles.
3. Inflammation of the iris, ciliary body, and cornea.
4. Cataract.
5. Disease of the retina and vitreous.
6. Disease of the optic nerve.

### FAILURE OF ACCOMMODATION; MYDRIASIS.

Failure of accommodation is, according to the majority of writers, one of the most common ocular symptoms in diabetes. The condition is generally one of limitation,—*i.e.*, of diminution in the range of accommodation, and seldom leads to complete loss of this power. Von Graefe[1] was the first to recognize this symptom in diabetics, and since his time numerous confirmatory observations have been recorded (Nagel, Seegen, Rosenstein, and others). It is met with in mild as well as in severe cases, and may be among the earliest evidences of disease. The onset, and especially the steady increase of presbyopia at an unusually early age, should excite suspicion of some general malady, and one of the most probable is diabetes. A case of Foerster's is recorded in which the premature onset of presbyopia led to the examination of the urine and the discovery of glycosuria. A very similar instance was observed by Seegen.[2] His patient was a young woman in whom failure of accommodation was noted, and who also complained of general muscular fatigue. The urine was found to contain 0.3 per cent. of sugar.

[1] Von Graefe, Archiv für Ophthalmologie, 1858, Bd. iv. Abth. 2.

[2] Seegen, Der Diabetes Mellitus, 2d ed., Berlin, 1875.

In the majority of these cases no abnormality of the pupil in size or in reaction is present. In some instances, however, there is evidence of paralysis or paresis of the iris; bilateral and unilateral mydriasis have been noted, and loss of or deficiency of the pupillary reaction to light (Ogle [1] and Testelin [2]). Among one hundred and forty cases of diabetes given by Seegen,[3] pupillary anomalies were noted in three. In one of these, however, a tumor of the medulla oblongata was found on post-mortem examination.

These conditions, cycloplegia and iridoplegia, have been variously explained. Foerster considered that the weakness of accommodation was part of the general muscular enfeeblement so usual in diabetics. Jacobson thought it due to peripheral neuritis, or to hemorrhage, while Berger and others maintain that these, as well as the extra-ocular paralyses, are toxic in origin. As evidence in favor of this view, Berger [4] adduces the observation (made by other writers also) that diminution of the glycosuria under treatment is followed by improvement in, or disappearance of, the intra-ocular paralysis.

### ALTERATION IN THE STATIC REFRACTION OF THE EYE.

A case reported by Horner [5] (apparently the only record of the kind), of acquired hypermetropia in a female fifty-five years of age suffering from severe diabetes, is referred to by most writers. There was no visible change in the crystalline lens. Under treatment for the diabetic condition, the degree of hypermetropia became less by about two diopters, while the presbyopia remained unaltered in degree. Horner suggested that the hypermetropia was due to a shortening of the eyeball by reason of the very large loss of fluid from its tissues.

A better known alteration in the refraction in diabetes is the development of myopia, to which attention has been especially drawn by Hirschberg,[6] who terms the condition "diabetic myopia." The onset of myopia in persons of fifty years of age or upward without discoverable abnormality of the lenses should always excite suspicion of diabetes. Hirschberg [7] gives three examples, in patients aged respectively sixty-three, fifty-two, and fifty. In all three there was reliable evidence of the recent onset of short sight, and in all the acuity of vision on the correction of the myopia was good. In cases of commencing diabetic cataract the refraction may become myopic. It is not unlikely, although unproved, that in the instances without visible change in the lens, the myopia is really due to an alteration in this structure,

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[1] Ogle, St. George's Hospital Reports, vol. i., 1866.

[2] Testelin, Annales d'Oculistique, t. xlix.

[3] Seegen, loc. cit.

[4] E. Berger, Les Maladies des Yeux dans leur Rapport avec la Pathologie générale, 1892.

[5] Horner, Klinische Monatsblätter für Augenheilkunde, 1873, S. 490.

[6] Centralblatt für Augenheilkunde, 1886, 1891.

[7] Ibid., 1890, S. 7.

whereby its refractive power is increased; the alternative hypothesis of elongation of the eyeball seems less probable.

### AFFECTIONS OF THE EXTRA-OCULAR MUSCLES.

The literature of diabetes contains many observations upon paralysis or paresis of the ocular muscles, and there is abundant authority to show that such complications of the disease are by no means infrequent. The exact relation these paralytic phenomena bear to the glycosuria has not been fully determined. Leber,[1] in reference to these ocular symptoms, suggests that the cases fall into two categories: first, those in which the paralysis is the result of diabetes, directly or indirectly; second, those in which the ocular paralysis is due to a cerebral lesion which also gives rise to the diabetes. He quotes seven cases belonging to his first group, and three cases which fall into his second group. Williamson,[2] in his recent work, includes ocular paralysis among the eye affections which are probably "merely accidental complications of diabetes;" this statement is, however, contrary to the views of most writers.

Paralysis of any of the ocular muscles may occur. It is generally stated that the muscles innervated by the oculo-motor (third) nerve are most frequently affected; but it is unusual to meet with paralysis of all the branches of this nerve. The levator palpebræ may be alone defective, producing unilateral ptosis. The sixth (abducens) or the fourth (trochlearis) nerve may be paralyzed, and characteristic diplopia result. Hirschberg states that the sixth nerve is most frequently attacked, and the writer's experience is in agreement with this statement. Paralysis of the facial nerve, and consequent loss of power in the orbicularis palpebrarum, may also be met with.

Paralysis of the ocular muscles is often slight and temporary, disappearing in the course of a few weeks; even if of longer duration, recovery usually ensues if the diabetic condition undergoes amelioration. According to Berger, however, ocular paralysis which comes on late in the disease is unlikely to be followed by recovery of the affected nerve. Recurrence of the paralysis is sometimes seen, or a second attack involving another muscle may be met with as in the case given below. The onset is frequently sudden, or at least the account given by patients is of the sudden development of diplopia and other symptoms.

These paralytic complications are met with in slight as well as in severe cases of diabetes; the majority of reported instances seem to have been in patients in whom the disease was not acute, and in whom treatment was followed by decided improvement. It is probably true that most of the cases of ocular paralysis recorded by ophthalmic surgeons have been in patients in whom the general symptoms were not severe, and who sought

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[1] Leber, loc. cit.

[2] Williamson, Diabetes Mellitus and its Treatment, London, 1898.

advice mainly or solely on account of the discomfort caused by the ocular trouble.

The following instances of ocular paralysis in diabetes may be mentioned.

A female, aged sixty-six, had suffered from diabetes "for some years," for which she had been under medical care. On September 1, 1891, she came under observation with a history of diplopia of three weeks' duration. There was well-marked paresis of the *right* external rectus muscle, with characteristic symptoms. Recovery ensued in two weeks—*i.e.*, about five weeks from the onset. At the time the ocular paresis came on, the urine had a specific gravity of 1032, and was reported to contain one-sixth of glycose, by the physician under whose care the woman had been. On August 17, 1893, the patient was again seen, with almost complete paralysis of the *left* external rectus of two weeks' duration. The urine had a specific gravity of 1018, and contained a very small quantity of sugar. The paralysis passed off in about five to six weeks from its onset. The patient was again under observation in 1897; the glycosuria persisted, but there had been no further ocular paresis, and the general health was well maintained.[1]

Gutmann[2] and Landesberg[3] each report a case of abducens paralysis in diabetes; in both instances recovery of the paralysis ensued.

James Anderson[4] recorded the case of a male aged forty-six, suffering from glycosuria, in whom there was paresis of the left internal and superior recti, which passed off under treatment in the course of three or four months, concomitantly with improvement in the general symptoms and diminution in the quantity of sugar excreted.

Seegen[5] reports, among other cases, one of bilateral ptosis in a female aged thirty-six, diabetic for nine months. The urine contained from three to nine per cent. of sugar, and although temporary improvement in the glycosuria resulted from treatment, the ptosis remained unaltered. The disease ended fatally, but no post-mortem examination was obtained.

Galezowski[6] records a case of paralysis of the third nerve in a diabetic in whom retinal hemorrhage was also present. Rolland[7] gives a case of complete right third nerve paralysis which recovered in six weeks under treatment.

Kwiatowski[8] reports a case under his observation in which the fourth nerve was paralyzed.

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[1] This patient was under the writer's observation.

[2] Gutmann, Centralblatt für Augenheilkunde, 1883, S. 299.

[3] Landesberg, Nagel, Jahresbericht, 1884, S. 323.

[4] Anderson, Ophthalmic Review, vol. viii., 1889, p. 38.

[5] Seegen, loc. cit., p. 299.

[6] Galezowski, Recueil d'Ophtalmologie, 1879, p. 75.

[7] Rolland, Archiv für Augenheilkunde, 1888, S. 257

[8] Kwiatowski, Nagel, Jahresbericht, 1879, S. 224.

The immediate causation of these ocular paralyses has been variously given. Knies[1] states that they may be due to nuclear and peripheral hemorrhage or to a peripheral neuritis. Berger considers they are induced by peripheral neuritis, which he thinks is toxic in origin. The most probable theory seems to be that of a peripheral neuritis, and this view would bring these ocular paralyses more into line with some of the other nervous manifestations of diabetes, to which peripheral neuritis is known to bear a causal relation. The apparent sudden onset of paralysis in some instances is, however, more suggestive of hemorrhage as an explanation.

### IRITIS AND KERATITIS IN DIABETES.

The occurrence of spontaneous iritis and iridocyclitis as an undoubted complication of diabetes was established mainly by the observations of Leber published in 1885.[2] A few isolated examples of iritis in diabetes had been previously recorded (Demarquay, Noyes, Galezowski, Condouris, and others), the earliest in 1863.[3] Since Leber's article appeared, and attention was specially directed to the subject, iritis as one of the ocular lesions in diabetes has become widely known, and is referred to by most writers. Judging from published statistics, it would appear to be one of the more common ocular complications. In thirty-nine cases of various ocular lesions in diabetics in Leber's clinique, there were nine cases of iritis. Hutchinson[4] states that in his experience iritis associated with glycosuria almost always occurs in patients who are also the subjects of gout. The liability of diabetics to iritis after operations on the eye, such as for cataract, is only too familiar to surgeons.

Diabetic iritis is not usually very severe. It is of the plastic type, and in a fair proportion of the cases is in appearance purulent,—*i.e.*, is accompanied by yellowish exudation into the anterior chamber. The hypopyon which forms is not usually large, nor are the inflammatory manifestations intense. The exudation, often fibrinous in character, may completely block the pupil by a membranous formation, or may lead to firm adhesions between the lens capsule and the iris. Leber found the pupil blocked by membrane in three out of nine cases of diabetic iritis. The exudation, however, frequently undergoes complete absorption under treatment; should this not occur, vision must necessarily be impaired. Signs of involvement of the ciliary portion of the uveal tract are present in a certain number of cases, followed by degenerative changes in the vitreous, from interference with nutrition.

In one of Leber's cases the iritis was of a serous character, followed

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[1] Knies, Die Beziehungen des Sehorgans zu den übrigen Krankheiten des Körpers, 1893.

[2] Leber, Archiv für Ophthalmologie, Bd. xxxi. Abth. 4, S. 184.

[3] Wiesinger, Archiv für Ophthalmologie, Bd. xxxi. Abth. 4, S. 209.

[4] Hutchinson, Transactions of the Ophthalmological Society of the United Kingdom, vol. v. p. 12.

the occurrence of hemorrhage into the vitreous, and resulted in glaucoma. The second eye of the same patient suffered from retinal hemorrhage, iritis, and glaucoma. Abadie has recorded a somewhat similar instance.

The iritis generally yields to treatment, local and general, but in some instances, notably in the post-operative cases, is intractable. Schirmer draws attention to the importance of examining the urine for sugar in cases of iritis in which treatment proves ineffective. The iritis is generally bilateral, but exceptions to this rule have been recorded (Schirmer).[1]

The occurrence of severe purulent destructive keratitis in patients suffering from diabetes has been observed in several instances. The keratitis, however, does not seem to differ materially from that which may occur in any prolonged wasting disease, and is merely a manifestation of the proneness of the corneal tissue to break down whenever its nutrition falls below a certain limit. Wiesinger[2] has collected and published a group of cases of keratitis in diabetes, but in several the relation of the diabetes to the corneal disease was very questionable; one patient had trigeminal paralysis, another was certainly syphilitic, and there were other elements of doubt.

## CATARACT IN DIABETES.

The occurrence of cataract in patients suffering from diabetes has been known for many years. It is one, perhaps the only one, of the ocular lesions met with in diabetics which is accepted by all writers as directly due to the glycosuric condition, although it is open to doubt whether all instances of cataract in elderly patients suffering from glycosuria should be considered as examples of diabetic cataract.

It is certainly one of the more frequent ocular complications, and by some authors is stated to be that most commonly met with. Von Graefe[3] estimated the proportion of cases of cataract in diabetics at twenty-five per cent. Galezowski[4] has recorded forty-six cases of cataract among one hundred and forty-four diabetics. Lagrange[5] gives particulars of fifty-two diabetic patients from Badal's clinique. Of these, one-fourth were affected by cataract. Koenig[6] found ten cases of cataract among fifty-six diabetics with ocular complications. Moore[7] found four cases of cataract in ninety-seven diabetics; Williamson,[8] in one hundred consecutive cases of diabetes under his own observation, found cataract in nine. Seegen,[9] on the other hand, had six cases of cataract in a total of one hundred and forty diabetics.

[1] Schirmer, Klinische Monatsblätter für Augenheilkunde, April, 1887.
[2] Wiesinger, Archiv für Ophthalmologie, Bd. xxxi. Abth. 4, S. 203.
[3] Von Graefe, Deutsche Klinik, 1859, S. 104.
[4] Galezowski, Nagel, Jahresbericht, 1883, S. 297.
[5] Lagrange, Archives d'Ophtalmologie, January–February, 1887.
[6] Koenig, Nagel, Jahresbericht, 1895.
[7] Moore, New York Medical Journal, March 31, 1888.
[8] Williamson, loc. cit.
[9] Seegen, loc. cit.

The lenticular trouble may develop at almost any age. One case at least is recorded in a girl aged eleven, and several in patients of from twelve to fifteen years of age. Of Williamson's nine cases referred to above, the youngest was twelve and the eldest fifty-nine years old. The lens opacity usually develops simultaneously in both eyes; a short interval between its onset in the two eyes may, however, be observed. At least one case of monocular cataract has been recorded (Seegen).

In its general appearance, cataract in diabetics presents nothing characteristic. Its causation cannot be determined with certainty without evidence of the existence of glycosuria. In the young, the cataract, which is of course of the soft variety, usually forms quickly, and may be complete in a few weeks. Its development is often accompanied by swelling of the lens, which becomes whitish, and diffusely opaque. As the condition is generally symmetrical, the onset of double, rapidly forming cataract in young or middle-aged patients should give rise to the suspicion of diabetes. In the old, the advance of the opacity is slower, and in its general appearance the cataract is similar to that occurring in elderly people without diabetes.

This ocular complication is met with not only in severe cases of diabetes in which there is much emaciation and enfeeblement, but also in those in which the general nutrition has suffered but little. This statement is especially, but not exclusively, applicable to the cases in patients beyond middle life. Seegen states that he has never found diabetic cataract in cases in which the urine contained only a small proportion of sugar. Other observers, however, have not had a similar experience. Thomas draws a subtle distinction between diabetic cataract and cataract occurring in diabetics, and suggests that the term "diabetic cataract" should be restricted to cases under fifty years of age, in which both lenses become affected simultaneously, or nearly so.

No one of the many theories advanced to explain the origin of diabetic cataract is wholly satisfactory. Among the suggested explanations are the presence of sugar in the lens acting as an irritant to its fibres, and leading to their degeneration; changes in the chemical constitution of the aqueous and of the vitreous reacting upon the crystalline lens; dehydration of the lens by the loss of fluid from the tissues generally; and, in a wider sense, interference with the nutrition of the lens induced by the general marasmus. To each of these hypotheses objections can be raised which render it untenable for all cases. Sugar has been detected in the transparent lenses in diabetes, as well as in those which were cataractous, and, according to Becker, cataract in one eye of a diabetic may contain sugar, and that in the fellow-eye be free from it. The presence of sugar in the aqueous and vitreous has also been demonstrated in diabetes. Deutschmann,[1] from the historical examination of four eyes from cases of diabetes, has described degenerative changes in the pigment epithelium of the iris and ciliary

[1] Deutschmann, Archiv für Ophthalmologie, Bd. xxxiii. Abth. 2.

portion of the uveal tract, and considers that these changes have an important bearing upon the occurrence of lenticular degeneration.

In one important feature, diabetic cataract differs notably from almost all other forms of cataract. Occasionally the lenticular opacity becomes less, and in some instances disappears altogether (Seegen,[1] Tannahill,[2] Moore[3]) if the general disease is treated and improvement ensues. In such instances it is evident that no serious degeneration of the lens fibres can have taken place. In reference to this point, it is interesting to note that cataract has been produced experimentally by the injection of a solution of sugar into the blood, and that the opacity of lenses thus induced disappears spontaneously. Some writers have looked upon this as proof that no comparison of the experimental and clinical cases is justifiable. The record of undoubted instances of spontaneous disappearance of lens opacity in diabetics, however, renders it probable that some of the cases in man are in many respects closely comparable to the experimental cases in animals, in which there is an excessive amount of sugar in the blood and tissues.

The practical question of the operative treatment of diabetic cataract does not come within the scope of this article, it is dealt with in the chapter on Diseases of the Lens. It will suffice to state that the suitability of such cases for operation will depend mainly (apart from evidence as to the integrity of the other ocular structures) upon the severity of the diabetes and the general condition of the patient. In the writer's experience, most of the cases of cataract in elderly diabetics are quite suitable for operative treatment, and the results are fairly satisfactory. In patients whose general health seems to be deteriorating as a result of the glycosuria, it is wise before operating to endeavor to obtain amelioration of the disease, and improvement in the general nutrition by a course of dietetic and medicinal treatment. Operation for the removal of diabetic cataract is said to be occasionally followed by diabetic coma.

## RETINAL DISEASE IN DIABETES.

The occurrence of retinitis in diabetes was first recorded by E. von Jaeger[4] in 1856; and in the same year Arlt referred to a case in which Treitz had found retinal disease in a diabetic. In 1858 Désmarres[5] (père) described cases of diabetes in which there were retinal lesions closely resembling those met with in Bright's disease, although the urine throughout contained only sugar. He suggested that possibly albuminuria had preceded the diabetes. The point raised by Désmarres was for a long time

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[1] Seegen, loc. cit.

[2] Tannahill, British Medical Journal, January 31, 1885. Quoted in Transactions of the Ophthalmological Society of the United Kingdom, vol. v. p. 107.

[3] Moore, loc. cit.

[4] E. von Jaeger, Beiträge zur Pathologie des Auges, S. 33, and Ophthalmologischen Handatlas, Vienna, 1869, Taf. XIII., Fig. 64.

[5] Désmarres, Traité des Maladies des Yeux, 2d éd., t. iii. p. 521.

unsettled. Much doubt was felt and expressed as to whether or no the association of glycosuria and retinal disease was more than a coincidence. Many considered that the retinitis was a variety of albuminuric retinitis, and this view received support from the fact that in not a few instances the urine contained both albumen and sugar. Noyes[1] in 1869 and Haltenhoff,[2] a few years later, showed that retinitis occurred in diabetes independently of any renal disease. Since then, and especially in recent years, the subject has attracted much attention, and there is now no room for doubt that, although in many cases the urine contains albumen as well as glycose, there is a retinitis in diabetes which is not albuminuric.

The frequency with which this ocular complication occurs is not easy to gauge. Williamson,[3] one of the most recent writers on diabetes, thinks that the frequency of retinitis in glycosuria has been over-estimated, and says that the statements in many text-books of medicine lead readers to suppose that retinal disease occurs in diabetes as commonly as in Bright's disease. "As a matter of fact, diabetic retinitis is very rare." His own experience is as follows: In one hundred cases of diabetes examined by him there were seven examples of retinitis; in three of these the urine contained much albumen as well as sugar; in two there was a trace of albumen but no other sign of nephritis; in two there was no albumen in the urine. Leber[4] speaks of retinitis as one of the less common eye affections in diabetes, "less frequent than optic nerve-disease, and much less frequent than cataract." Moore[5] found five cases of retinitis in ninety-seven diabetics. De Wecker[6] notes that retinitis in diabetes uncomplicated by albuminuria is very rare, and Seegen found but two cases in one hundred and forty diabetics.

In some statistics given by Lagrange, and already referred to, the proportion of cases of retinitis in diabetes is nearly equal to that of cataract. This would seem, by comparison with the records of other writers, to be too high a percentage. Hirschberg has stated that the association between glycosuria and retinitis is so close that the latter is rarely absent in cases in which glycosuria has existed for several years.

Retinitis is one of the late complications of diabetes, developing when the disease is advanced, and usually in cases in which polyuria and other symptoms have existed for a long time, perhaps for years. It is not met with, or but very rarely met with, in cases running an acute course, and is generally found in patients who are over forty-five years of age. The first recorded case, however, was in a male aged twenty-two, and of fourteen cases given by Leber, of which the age is specified, six were under forty-five years. The writer has not seen retinitis in a diabetic under fifty. The

[1] Noyes, Transactions of the American Ophthalmological Society, 1869.

[2] Haltenhoff, Zehender's Klinische Monatsblätter für Augenheilkunde, xi S. 291, 1873.

[3] Williamson, loc. cit.

[4] Leber, Graefe-Saemisch, Handbuch der gesammten Augenheilkunde, Bd. v. S. 595.

[5] Moore, loc. cit.

[6] Traité complet, de Wecker et Landolt, vol. iv.

## PLATE VI.

Retinitis in glycosuria. (Nettleship, Transactions of the Ophthalmological Society of the United Kingdom, 1886, vol. vi.) Ophthalmoscopic drawing of the right eye of a man aged fifty, suffering from chronic glycosuria. V = 18 Jaeger, badly. The optic disk and retinal vessels are healthy looking. A large number of white patches and spots are scattered over the central region of the fundus, and some similar spots are seen in the nasal part. At the macula is a larger, dense, irregular mass of deposit. The urine had a specific gravity varying between 1030 and 1040; it contained a large quantity of sugar, but no albumen.

fact that retinitis is so rare in the more severe forms of diabetes and in young patients suggests that there is some factor necessary for its production other than the diabetic condition of the blood (Williamson).

The patients in whom retinitis develops are not always seriously ill. Some of them come under observation only because of the failure of vision, although inquiry usually discloses symptoms pointing to a prolonged period of glycosuria. One of the most marked cases of glycosuric retinitis the writer has seen occurred in a female over sixty years of age, who came for advice only because she was unable to see to do needlework. The disease commonly affects both eyes, although not always to the same degree. The defects of sight may vary within wide limits, depending upon the extent and locality of the changes in the retina, and also, in some instances, upon the presence of the secondary changes in the vitreous which are so frequently present.

The retinal disease is usually chronic, and may undergo but little alteration during a period of many weeks. On the other hand, considerable change in the ophthalmoscopic appearances may result from repeated hemorrhage from the retinal vessels. Even if improvement occur under treatment the retinal disease is very prone to relapse. The description of the ophthalmoscopic appearances of glycosuric retinitis and of the variations which the disease assumes, as well as its morbid anatomy, are dealt with in a previous part of this system (vol. iii.)[1] A brief reference to some of the ophthalmoscopic features of the disease may be made here, and the accompanying drawing (Plate VI.)[2] is given as illustrative of one of the varieties of diabetic retinitis. The retinal changes in diabetes most closely resemble those met with in albuminuria, and sometimes in pernicious anæmia. The practical difficulty in diagnosis is as between glycosuric and albuminuric retinitis. Among the most noteworthy differences are the following: In diabetic retinitis the whitish patches in the retina are larger, they are not so generally confined to the macular region, and they exhibit little or no tendency to the fan-shaped or stellate arrangement so usual in albuminuric cases. The optic papilla seldom shows signs of inflammation. Hemorrhage in the retina, though occasionally absent, is often abundant, and the extravasations are in circular spots or points or irregular patches, seldom flame-shaped, as in albuminuric cases. The presence of hemorrhage or the results of hemorrhage in the vitreous, which is rare in albuminuric, is very frequent in diabetic retinitis. These hemorrhages, which are derived from retinal vessels (Leber and Nettleship), and which tend to recur, are of much importance in the ophthalmoscopic diagnosis of glycosuric retinitis. If looked for, opacity of the vitreous of this kind will be found in a large proportion of cases.

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[1] The best description of the morbid anatomy of glycosuric retinitis of which the writer is cognizant is in an article by S. Mackenzie and Nettleship, Ophthalmic Hospital Reports, vol. ix. p. 134.

[2] Copied from Nettleship's colored plate in Transactions of the Ophthalmological Society of the United Kingdom, vol. vi. Plate II.

The prognostic significance of retinitis in diabetes is a matter of much importance. All authors are agreed that its occurrence is a bad omen, but some difference of opinion exists as to the seriousness of it (Leber, Saundby, Williamson). There can be little doubt that as retinitis usually occurs late in the course of diabetes, and the changes in the retina are indicative of advanced degenerative processes in the tissues, this complication should be regarded as very grave, and as giving to a case of otherwise mild diabetes a very serious aspect. Statistics are wanting as to the duration of life in diabetics after the onset of retinitis.

### DISEASE OF THE OPTIC NERVE IN DIABETES.

That the optic nerve in diabetes is sometimes the seat of degenerative changes and consequent impairment of function is undoubted. But no little confusion has arisen regarding this subject, and for various reasons. In the first place, many of the older cases in literature are almost certainly, and some are certainly, cases in which the optic nerve disease was directly due to a cerebral lesion, and not the result of glycosuria. Secondly, it seems more than probable that some of the cases formerly described as examples of amblyopia without ophthalmoscopic changes would now be regarded as instances of partial degeneration of the optic nerve with slight but recognizable signs of disease.

The frequency of optic nerve disease in diabetes is not easy to determine. Of fifty cases with ocular symptoms, observed by Leber,[1] there were fourteen—*i.e.*, twenty-eight per cent.—with optic nerve trouble. In one hundred and forty diabetics with eye affection, Schmidt-Rimpler[2] found thirty-four in whom there was optic nerve disease.

"Diabetic amblyopia" may be conveniently referred to here. Although in the majority of instances the amblyopia is probably due to an optic nerve lesion, there are some cases in which a considerable degree of failure of sight occurs without any discoverable change in the fundus oculi. In these instances, or some of them, the amblyopia is toxic in origin, due to poisoning of the cerebral centres, and is comparable to the amaurosis of uræmia; like the latter, the blindness may pass off under treatment. Anderson[3] pointed out that the sudden onset of amblyopia in diabetes "without ophthalmoscopic explanation probably presages the near onset of coma, having in this relation much the same significance as the amblyopia of uræmia." It usually occurs early in the disease, and not infrequently soon after the adoption of strict antidiabetic treatment.

More frequent than this affection of sight are those cases in which there is a definite lesion of the optic nerve. Leber, in his most valuable article so often referred to, gives three headings for cases of optic nerve

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[1] Leber, Bericht der ophthalmologischen Gesellschaft, Heidelberg, 1896, S. 104.

[2] Schmidt-Rimpler, Bericht der ophthalmologischen Gesellschaft, Heidelberg, 1896, S. 99.

[3] James Anderson, Ophthalmic Review, vol. viii., 1889.

disease in diabetes. First, amblyopia without ophthalmoscopic changes, with and without limitation of the field of vision; secondly, optic nerve atrophy; thirdly, hemianopia. It will be convenient to deal with these groups in a reverse order.

The evidence concerning hemianopia in diabetes is scanty, very few cases having been recorded; there is no reasonable doubt that in the reported cases the symptom was caused by localized intracranial diseases and was not directly due to the glycosuria.

Atrophy of the optic nerve in diabetes, in which well-marked changes are visible in the optic disk, is met with either in the primary form, or as atrophy occurring coincidently with, and probably secondarily to, diabetic retinitis. In the primary form, visual acuity is much lowered, the failure may be rapidly progressive, and sight may be completely lost. The field of vision shows well-marked contraction, sometimes concentric in character, sometimes very irregular in outline. The ophthalmoscopic appearances of the optic disk in this variety of disease do not differ from those of simple atrophy from other causes. This complication of diabetes is evidently a very unusual one. The cases on record in which a simple optic nerve atrophy was present are few in number, and in several of them it is questionable if the optic nerve lesion should be considered a result of the glycosuria. In two instances the results of post-mortem examination have been recorded.[1] In one of these, a tumor was found pressing on the optic chiasma; in the other no cerebral lesion was discovered which would account for the optic atrophy, but there was advanced degeneration of the kidneys.

Amblyopia without (gross) ophthalmoscopic changes, in which there is greater or less defect in the central part of the field of vision, with little or no limitation of the periphery, has been described by many writers, and widely recognized.[2] Considerable difference of opinion has existed, and may still exist, in reference to some features of this complication. In recent years, however, several important contributions to our knowledge have dissipated much of the doubt.

In this form of amblyopia, the defect of central vision may vary from the slightest diminution in acuity to an absolute loss. There is, in addition to lowering of the visual acuity, a central scotoma for color. Red and green are the colors, the recognition of which is most commonly deficient, and appreciation of blue has also occasionally been noted as defective. The scotoma is not always easy of detection. It may involve a very small portion of the field, and may be very relative and paracentral. In well-marked instances it occupies an area which extends from the centre outward as far as, and often beyond, the blind spot. The defect is generally symmetrical;

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[1] *Vide* Archiv für Ophthalmologie. Bd. xxi. Abth. 3, 298, 299.

[2] Leber, Graefe-Saemisch, Handbuch der gesammten Augenheilkunde, vol. v.; Lecorché, Gazette hebdomadaire de Médecine et de Clinique, November, 1861; Begbie, Edinburgh Medical Journal, June, 1861.

Galezowski[1] makes the statement that one eye only may be affected, but this has not been confirmed by other writers.

Amblyopia of this kind may occur in diabetics whose general health is not much undermined, but it is more frequently found in cases in which considerable loss of strength and vigor has resulted. It is rare in patients under thirty, and in most of the published cases the age was over forty. Bresgen[2] records a case in a man twenty-four years of age, and Schmidt-Rimpler[3] and Edmunds and Lawford[4] two cases in men aged twenty-nine. The onset of symptoms is generally rather abrupt, and the amblyopia may rapidly increase to a considerable degree, and then become stationary, or may "vary according to the diabetic condition" (Moore and Lecorché); complete loss of sight is a very rare result.

Records of a very considerable number of cases of central amblyopia in diabetes can be found in ophthalmic literature. In all the earlier cases the condition is described as one of amblyopia without ophthalmoscopic changes. In the more recent records of the same class of cases, slight but recognizable ophthalmoscopic changes are noted. In reference to this point, Schmidt-Rimpler[5] writes: "The optic disk shows more frequently than is supposed, slight, but undoubted pathological changes." These changes consist, in some instances, of hyperæmia of the papilla, and some little connective-tissue disturbance on its surface; in the larger number, of pallor of the temporal part of the disk.

These cases bear a close resemblance, in nearly all respects, to certain well-known forms of toxic amblyopia, and especially to that induced by tobacco, or tobacco and alcohol. In both classes there is defect of vision of very varying degree, in both a central scotoma for form and color, but (generally) no limitation of the field of vision; and in both ophthalmoscopic changes are either absent or are so slight that they may easily be overlooked. Galezowski[6] appears to have been the first to direct attention to the similarity of central amblyopia in diabetes and that caused by alcohol and tobacco. This was in 1879. About the same time, and during the next few years, several well-observed instances of central amblyopia in diabetic patients were placed on record, and the question as to the exact relation existing between the glycosuria and the visual defect came to be critically discussed. In nearly all instances the patients were males, and were smokers. Some were also given to excess in alcohol. Hence arose the difficulty, and in some instances the impossibility, of determining to what extent the visual defect was due to the diabetic condition, and to what

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[1] Galezowski, Recueil d'Ophtalmologie, 1879.

[2] Bresgen, Centralblatt für Augenheilkunde, 1881.

[3] Schmidt-Rimpler, loc. cit.

[4] Edmunds and Lawford, Transactions of the Ophthalmological Society of the United Kingdom, vol. iii., 1883.

[5] Schmidt-Rimpler, loc. cit.

[6] Galezowski, loc. cit.

extent it was caused by tobacco or alcohol. And some observers hold, or have held, the opinion that these cases are in reality instances of tobacco amblyopia in diabetics. That this view is correct in regard to a considerable proportion of the cases cannot be doubted (Leber, Nettleship, Lawford). Leber believes that the existence of diabetes renders the individual more susceptible to the toxic effect of tobacco than he would otherwise be. But there is some evidence, and it is apparently slowly accumulating, that central amblyopia may be induced by diabetes without the aid of tobacco or other extraneous poison. Four cases are on record of central amblyopia in women suffering from diabetes;[1] one reported by Nettleship[2] in 1881, and one by Samuel[3] from Hirschberg's clinique in 1882, and two by Moore[4] in 1888. Nettleship speaks of his case as inconclusive, owing to the fact that the defect of vision was very slight and the patient only examined on one occasion. Hirschberg refers to Samuel's case as undoubted.

Nettleship's patient was a thin, feeble woman, aged thirty, with diabetes of about seven months' duration. Vision, when the patient was examined, was, with each eye, 6/6 slowly and partially, with correction of some hypermetropia. She could not read the smallest print easily with any lens. There was a small central scotoma for red and green, but no limitation of the field of vision. There were no ophthalmoscopic changes. Samuel's case was a female aged fifty-two, who had had iritis in both eyes, leading to occlusion of pupils, for which iridect my had been performed. A central scotoma was pre-ent in each, but the fields of vision were almost full. Vision was 15/200, and with + 5.0 D. the patient could read Snellen 13. The urine contained 4.5 per cent. of sugar, the general symptoms of diabetes were slight, and the patient's health was but little impaired. Moore's cases were, briefly, as follows: (1) Female, forty-nine years of age, with a history of diabetes for six months. Rapid failure of vision for one week before she came under observation. Urine contained a large quantity of sugar. Vision in each eye 20/200; no limitation of field, but a large central scotoma for red and green. No ophthalmoscopic changes except doubtful pallor of temporal part of optic disk. Vision remained stationary. Patient died two years later. (2) Female, aged fifty-one; diabetes for some months. Urine contained sugar in abundance. Failure of vision in right eye and subsequently in left. Vision 20/50 in each eye, not improved by glasses. No abnormal appearances in the fundi except a slight dusky hue of the optic disks. No contraction of fields. Large central scotoma for green; relative scotoma for red. Six months later vision was 20/200 in

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[1] With the exception of a case reported by Désmarres (Traité des Maladies des Yeux, 1858), the notes of which are incomplete, the four cases given are the only instances of central amblyopia in female diabetics of which the writer has found records.

[2] Nettleship, Transactions of the Ophthalmological Society of the United Kingdom, vol. i. p. 127, and vol. iii. p. 165.

[3] Samuel, Centralblatt für Augenheilkunde, July, 1882.

[4] Moore, New York Medical Journal, March 31, 1888.

each. Patient died of gangrene of foot twelve months after she was first seen by Moore. In all these cases in women, tobacco as a cause of the amblyopia was excluded. In Hirschberg's case it is also stated that there had been no excess in alcohol.

Eales[1] in 1881 reported a case of central amblyopia, with color scotoma, in a male diabetic, who *probably* did not smoke.

A more recent case, reported by Schmidt-Rimpler,[2] is important, in that causes of central defect of vision other than diabetes can be almost certainly excluded. The patient was a man twenty-nine years of age, who had well-marked central amblyopia (V. R. $= \frac{1}{6}$; L. $= \frac{1}{4}$), with absolute color scotoma, and full fields of vision, and who was suffering from severe diabetes; the urine containing five per cent. of sugar, but no albumen. The general symptoms (polyuria, emaciation, etc.) had been noticed for nearly a year, and failure of vision had soon followed. The patient had smoked but little; often for weeks at a time he would not touch tobacco. He took alcohol in very moderate quantity (these statements were fully confirmed by his wife). Coma supervened, and the man died a few days after Schmidt-Rimpler saw him.

These six cases, if not proof of the occurrence of central amblyopia of purely diabetic origin, are at least valuable evidence in favor of it.

In several instances, the optic nerves of patients dying of diabetes, and who during life were amblyopic, have been examined microscopically. Nettleship and Edmunds[3] (1881), Edmunds and Lawford[4] (1883), Fraser and Bruce[5] (1896), and Schmidt-Rimpler[6] (1896), have recorded the results of microscopic examination of the optic nerves in diabetes. The condition found is one of degeneration of nerve-bundles in a tract which runs axially through the orbital portion of the nerve to the chiasma. The position of this degenerate tract relative to the centre of the nerve varies at different levels. It involves the bundles of fibres whose ultimate distribution is chiefly to that part of the retina between the optic disk and the macula, and which are known as the "papillo-macular bundles" (Henschen). For a more detailed description, the reader is referred to the original papers. Fraser and Bruce's case is of more than usual interest, in that the patient suffered from symptoms indicative of peripheral neuritis, and that degenerative changes were present in spinal nerves, similar to those in the optic nerve. This patient had been a heavy smoker, but temperate in the use of alcohol. All the cases referred to had during life central defect of vision, and all were smokers except Schmidt-Rimpler's patient. His case,

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1 Eales, The Lancet, 1881, vol. ii. p. 200.

2 Schmidt-Rimpler, loc. cit.

3 Nettleship and Edmunds, Transactions of the Ophthalmological Society of the United Kingdom, vol i. p 124 (with plate).

4 Edmunds and Lawford, Ibid., vol. iii. p. 160 (with plate).

5 Fraser and Bruce, Edinburgh Medical Journal, October, 1896 (with plate).

6 Schmidt-Rimpler, loc. cit.

in its clinical aspect, and in the microscopic changes in the optic nerve, appears to coincide exactly with the other three, and as in it the effect of tobacco and alcohol could be almost certainly excluded, there seems reasonable ground for believing that diabetes may lead to an axial neuritis similar in its histological characters to that induced by toxic agents such as tobacco and alcohol.

The prognosis in cases of central amblyopia in diabetes appears to be very doubtful. Leber speaks rather hopefully on this point, and mentions the marked improvement which may follow antidiabetic treatment. Other writers consider the prognosis far from encouraging, especially in cases in which there is an absolute scotoma. It is not improbable that in some instances the degeneration, which is at first axial and limited in the optic nerve, may extend to adjoining portions, or that the degeneration of nerve-fibres may progress to a point beyond which recovery is impossible. The clinical evidence, however, favors the view that extension of the disease in the optic nerve beyond the papillo-macular bundles rarely if ever occurs.

### III. DIABETES INSIPIDUS; POLYURIA; POLYDIPSIA.

The two chief symptoms of this malady are great increase in the quantity of urine and distressing thirst; hence the terms "polyuria" and "polydipsia"; the latter being given in the belief that the thirst and the consequent consumption of large quantities of fluid were the essential cause of the increased flow of urine. It is now known, however, that there is no constant relation between the amount of urine passed and the amount of fluid imbibed. A patient suffering from diabetes insipidus will pass more urine than a healthy person who drinks an equal quantity of fluid.

The secretion of urine in diabetes insipidus may amount to twenty or thirty pints in twenty-four hours, the specific gravity being from 1003 to 1007. The percentage of solids is low, but the total amount of urea excreted appears to be augmented rather than diminished. There is an absence of albumen and glycose.

This disorder has to be distinguished from saccharine diabetes on the one hand, and organic disease of the kidneys, with polyuria as a symptom, on the other. In the former case there is no difficulty; the presence or absence of sugar in the urine at once decides the question. In the latter case, however, the diagnosis, especially in elder people, is not so simple. Renal cirrhosis may be very insidious in onset, albumen in the urine may be small in quantity or not constantly present, and an increased flow of urine may be the most noticeable symptom. In such instances, other aids to diagnosis than the examination of the urine will prevent mistakes. Careful investigation of the vascular system in cases of renal cirrhosis will reveal increased arterial tension, and, in all probability, evidence of degenerate vessels. In diabetes insipidus, the arterial tension is lowered rather than excessive (Fagge).

The records of ocular lesions in diabetes insipidus are not numerous.

A careful study of the published cases seems to indicate clearly that, in the majority of instances, the ocular symptoms are "connected with the cause rather than with the condition of polyuria" (Gowers). Such is almost certainly true of the cases of optic neuritis reported by van der Heyden,[1] unilateral optic nerve atrophy published by Laycock,[2] temporal hemianopsia and paralysis of sixth nerve recorded by von Graefe,[3] and temporal hemianopsia reported by David.[4] In such cases, with scarcely any doubt, the cerebral lesion which gives rise to the ocular complication is also responsible for the polyuria. In no one of these cases has the writer been able to find a record of a post-mortem examination. David's patient died with symptoms of gross intracranial disease, but no autopsy was made.

Two cases of diabetes insipidus have been recorded by Galezowski,[5] in which he found retinal hemorrhages. The extravasations were small, situated peripherally, and gave rise to no subjective symptoms. No note is given of the appearance of the retinal vessels, but it is stated that there was no alteration in the papilla, or in any tunic other than the retina. Gayet[6] has recorded a case of paralysis of the right sixth nerve occurring suddenly in a male aged twenty-eight suffering from diabetes insipidus. The urine was limpid, clear, free from albumen, sugar, or phosphates. Improvement of all the symptoms occurred under treatment. This case and Galezowski's two cases seem to merit consideration as true ocular complications of the disease.

## IV. OCULAR LESIONS IN OTHER ABNORMAL CONDITIONS OF THE URINE.

Scattered through medical literature, a few records can be found of cases in which ocular disease was associated with abnormal conditions of the urine other than those of albuminuria, glycosuria, and diabetes insipidus. The reports of some of these cases are incomplete, and the total number is so small that no dogmatic statements can be safely made as to the relation between the ocular symptoms and the condition of the urine. Such associations are, however, worth mentioning, and should be borne in mind in the hope that further observation may clear up the doubtful points.

In *oxaluria*, or that condition in which the urine contains oxalate of lime in excess, disease of the retina and vitreous has been noted. Cases of this description have been reported by Mackenzie[7] and Leber;[8] and so

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1 Van der Heyden, Nagel's Jahresbericht, 1875, S. 191.

2 Laycock, The Lancet, 1875, vol. ii. p. 242.

3 Von Graefe, Zehender's Klinische Monatsblätter für Augenheilkunde, iii., 1865, S. 268.

4 David, Nagel's Jahresbericht, 1889, p. 492.

5 Galezowski, Traité des Maladies des Yeux, 1871, p. 601; and Traité iconographique d'Ophtalmoscopie, 2d edit., 1886, Plate V.

6 Gayet, Recueil d'Ophtalmologie, 1876, p. 171.

7 Mackenzie, Annales d'Oculistique, t. liii. p. 248, 1865.

8 Leber, Graefe-Saemisch, Handbuch der gesammten Augenheilkunde, Bd. v. S. 597.

long ago as 1850, Bouchardat drew attention to the coincidence of visual troubles and oxaluria. In Mackenzie's and Leber's cases there was retinal disease of a hemorrhagic type, with the formation of cicatricial bands and new vessels in the vitreous. Both observers report improvement in the ocular condition under treatment directed to the abnormality of the urine.

In *phosphaturia*, ocular symptoms have been noted, and along with other ill-defined, chiefly nervous, troubles have been attributed to the abnormal condition of the urine, or, more correctly, to that disturbance of chemical processes which leads to the excess of phosphates in the urine. Phosphaturia as a symptom appears in many different conditions of disease, and our knowledge of its pathology and significance is but meagre. It may be in this, as in other abnormal states of the urine, that the ocular and other symptoms are merely associations of the phosphaturia, and that all are due to a common cause. The close, though unexplained, relation between the conditions of phosphaturia and glycosuria should be borne in mind.[1] Hansell[2] has recorded the case of a man aged thirty-nine, in whom he found retinal changes resembling those of albuminuric retinitis, although not quite characteristic. No subjective symptoms were complained of except failure of vision. The urine was abnormally abundant (seventy-nine ounces in twenty-four hours) and contained an excess of phosphates, but no albumen or sugar, on repeated examination. Trousseau[3] reports several cases of retinal hemorrhage which he found associated with phosphatic urine.

The *uric acid diathesis* has been credited with at least a share in the production of some forms of ocular disease, but it is needful to remember that excess of lithic acid in the urine is usually merely a symptom of some general malady, and that the coincident ocular lesions may be only another indication of the morbid condition. Among the few reports of cases may be mentioned those of Bergmeister,[4] who recorded four instances of posterior polar cataract in patients whose urine contained an excessive quantity of uric acid.

## OCULAR LESIONS IN DISEASES OF THE SKIN.

The ocular lesions met with in cases of skin-disease are, in most instances, merely an extension of the cutaneous affection to the tissues of the eyelids and globe. In most of the recognized affections involving the integument of the face and spreading to the eyelids, the identification of the disease in the latter situation presents little difficulty, although its characters may undergo some modification by reason of the peculiar anatomical conditions in the lids. The extremely thin skin, the loose subcutaneous cellu-

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[1] Anderson, Ophthalmic Review, vol. viii., 1889; Tessier, Du diabète phosphatique, Paris, 1896.

[2] Hansell, The Philadelphia Polyclinic, January 21, 1897.

[3] A. Trousseau, Le Bulletin médical, April, 1897.

[4] Bergmeister, Nagel's Jahresbericht, 1894, S. 528.

lar tissue, and the abundance and large size of glandular structures, all tend to modify the appearances of inflammatory affections when involving these parts. In cases in which disease of the skin extends to the palpebral or ocular conjunctiva, the appearances may be altered so as to give rise to some difficulty in diagnosis.

The involvement of the skin of the eyelids by eczema, impetigo, and other common cutaneous diseases need only be referred to here. These conditions are dealt with in an earlier section of this work. The same remarks apply in great measure to the more destructive lesions, such as lupus (tubercle), and various forms of epithelioma, which, commencing in the adjacent skin, attack in their course the palpebral and, subsequently, the ocular tissues.[1]

There are, however, certain skin-diseases in the course of which lesions of some of the ocular structures arise which cannot be regarded as mere extensions, by continuity, of disease from neighboring parts. Again, some observers have noted serious disease of the eye (*e.g.*, cataract) in association with severe skin affections, and have considered the former as secondary to and induced by the latter. In the first category, the ocular lesion, in at least some of its clinical characters, resembles the lesion in the skin. In the second group there is little or no resemblance. Among the most remarkable of the ocular affections in disease of the skin is that met with in

*Pemphigus.*—Much difference of opinion has been expressed concerning the relationship between pemphigus and a peculiar intractable and destructive affection of the conjunctiva. The latter has been described as *pemphigus of the conjunctiva* and as *essential shrinking of the conjunctiva;* an account of its clinical features is given in the article on diseases of the conjunctiva, vol. iii. p. 226. The difficulties in determining the exact nature of the eye affection are considerable, and are not lessened by the fact that dermatologists are by no means agreed as to the diagnosis between pemphigus and some other bullous eruptions on the skin. The development of the bullæ of pemphigus on mucous membranes other than the conjunctiva is well known. In the mouth and pharynx they are not uncommon; they do not, however, exert so baneful an influence here as in the conjunctiva, and although a certain amount of contraction of the buccal mucous membrane may result, with the formation of bandlike adhesions, the disease is not followed by the very extensive shrinking of tissue and destruction of its secretory powers that is seen when the conjunctiva is attacked. The very different result (as regards scarring) of pemphigus when affecting the skin and when involving mucous membranes is noteworthy.

That bullæ are found on the conjunctiva and cornea in many of these cases, and that they are associated with a bullous eruption on the skin, or on the buccal mucous membrane in some, is sufficiently attested by pub-

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[1] The ocular complications of erysipelas and of herpes zoster are described in other sections.

lished records. There are, however, reports of cases in which no vesicles were seen on the conjunctiva or elsewhere, but in which the atrophic changes in the conjunctiva and cornea were well marked. Under such circumstances, some observers have reasonably doubted whether the diagnosis of pemphigus was justifiable. The opinion seems to be gaining ground, however, that the term "pemphigus" is the correct one to apply to the conjunctival and corneal condition originally described by White-Cooper[1] under this name.

Statistics (Wilson, Kaposi, and others) show that pemphigus is one of the rare diseases of the skin. It is therefore not surprising to find that pemphigus of the conjunctiva is an uncommon affection. It may occur at any age; White-Cooper's patient was a woman aged twenty-four, and the next two recorded cases (de Wecker,[2] Hardy[3]) were seventy-one and sixty-eight years of age respectively. In many of the published cases the disease occurred in infancy or childhood. In twenty-eight cases collected by Morris and Roberts,[4] the youngest specified age was four, and the oldest seventy-six. Three cases occurred in "small children." The average age in twenty-five cases was forty-one. The recorded cases of pemphigus of the conjunctiva do not agree with the facts as regards age given by writers on diseases of the skin, according to whom pemphigus is much more common in children.

The ocular affection is usually bilateral, though often unequal in degree in the two eyes. In its tendency to symmetry, it contrasts noticeably with the skin lesions. There seems to be no constant relation between the duration of the skin affection and the development of the eye lesions. In some instances only a few weeks have elapsed from the first appearance of bullæ on the skin until the conjunctiva became implicated. In others the interval has been measured by months (Hardy) or even years. It has been stated that pemphigus may primarily affect the conjunctiva, and in the twenty-eight cases collected by Morris, the disease was said to have begun in the conjunctiva in eight (28.5 per cent.). It seems probable that in some such instances the cutaneous lesions have been overlooked, although really preceding the ocular complication. This is one of the many points upon which information is wanted in regard to this interesting and serious malady. To be of value, the information must be obtained from careful examination and observation.

The conjunctival lesions are met with in cases in which the skin affection is chronic, and not severe. So far as the writer is aware, no instances have been recorded of conjunctival lesions in acute pemphigus. The course of the ocular disease, like that of the skin, may be very chronic. Samelsohn

[1] White-Cooper, Royal London Ophthalmic Hospital Reports, vol. i., 1858, p. 155.
[2] De Wecker, Klinische Monatsblätter für Augenheilkunde, 1858.
[3] Hardy, Traité complet, de Wecker et Landolt, vol. i. p. 438.
[4] Morris and Roberts, Monatschrift für praktische Dermatologie, May, 1889. These tables included all published cases up to date.

observed a case in which bullæ repeatedly developed on the conjunctival surface of the lids during seven years, and but little atrophy resulted. In other instances, however, a much shorter period sufficed for extensive destruction of tissue. In a case recorded by Silcock,[1] one eye was destroyed between six and seven weeks after the patient came under observation, and the other was seriously damaged. Suppuration of the cornea has occurred in a few instances, and has led to rapid destruction of the eye.

The outlook, when once pemphigus attacks the conjunctiva, is extremely unfavorable; the morbid process nearly always progresses till sight is entirely destroyed. Only three reported cases of recovery have come under the writer's notice, two by Samelsohn,[2] and one by Marcus Gunn.[3]

In addition to those mentioned in the text, notes of cases of pemphigus of the conjunctiva, and references, will be found in the Transactions of the Ophthalmological Society of the United Kingdom, 1886, 1893–97, and in a paper by Cohn[4] and in one by Tilley.[5] Reports of the microscopic examination of the diseased conjunctiva are given by Sattler, Gelpke, and Bäumler.[6]

In two of the more common diseases of the skin, erythema and psoriasis, ocular lesions, not from extension, have been described. Our knowledge regarding these lesions and the relation they bear to the cutaneous affection is too limited to merit anything more than a brief statement of the observations which have been made.

*Erythema.*—Several cases have been recorded[7] in which, during an attack of erythema multiforme, large papillary and in some instances vesicular elevations developed on the ocular conjunctiva, usually on the nasal part. They give rise to very few subjective symptoms, are generally bilateral, and disappear in the course of a week or ten days, leaving the tissues unaltered in appearance.

*Psoriasis.*—It has been stated that in this disease of the skin the conjunctiva is sometimes affected.[8] Small foci of inflammation develop, which lead to the formation of ulcers. Iritis has also been noted. There is, however, insufficient evidence to establish a causal connection between the disorder of the skin and the inflammation of the conjunctiva and iris.

In the second group of cases (that in which the ocular lesion is in no way similar to the skin-disease) the association may be purely accidental.

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1 Silcock, Transactions of the Ophthalmological Society of the United Kingdom, vol. xvii., 1897.

2 Samelsohn, Bericht der Ophthalmologischen Gesellschaft, Heidelberg, 1879.

3 Marcus Gunn, Transactions of the Ophthalmological Society of the United Kingdom, vol. xv., 1895.

4 Breslauer aerztliche Zeitschrift, 1885, Nr. 10.

5 American Journal of Ophthalmology, June, 1887.

6 Klinische Monatsblätter für Augenheilkunde, xvii. und xxiii.

7 Beaudonnet, Nagel's Jahresbericht, 1894, S. 582; Terson, Annales d'Oculistique, t. cxiv. p. 304, 1895.

8 Joueix, Nagel's Jahresbericht, 1896.

Mooren,[1] Foerster,[2] Nieden,[3] and Rothmund[4] have published observations concerning the occurrence of cataract in patients suffering from chronic inflammatory skin-disease; and both Mooren and Nieden hold that prolonged affection of the skin definitely predisposes to the formation of cataract. Foerster's opinion was that the chronic skin-disease induced a general cachexia, of which the cataract was merely one of the manifestations.

The cases recorded by Nieden and Rothmund are of interest even if the causal relation of the cutaneous malady be considered doubtful. Nieden's case was that of a girl in whom facial telangiectasis developed at the age of twenty-two. At the same time opacities formed in the cortical layers of the left crystalline lens, and posterior polar cataract in the right. Rothmund's cases occurred in the children of three families[5] living in adjacent villages in Austria. There were fourteen children, of whom seven were the subjects of a very unusual form of skin-disease characterized by a fatty degeneration of the Malpighian layer and of the papillæ, and subsequent atrophy of the epidermis. This disease attacked the skin of the face and arms between three and six months of age. A few years later—*i.e.*, between the third and sixth year—cataract began to develop, and eventually both lenses became completely opaque. Of the seven children affected with the skin-disease, five had double cataract, the remaining two were under two years of age at the time of the report.

Mooren and others have stated that chronic skin eruptions may be accompanied by optic neuritis and retinitis. These statements, however, require confirmation before the association can be considered as anything more than a coincidence.

## OCULAR LESIONS IN DISEASES OF THE DIGESTIVE ORGANS.

The ocular lesions described as occurring in the course of the various disorders of the digestive organs (stomach, intestines, liver, etc.) must in many instances be regarded as having but an indirect connection with the gastric or other affection. It is not very often that a causal relation between the general disorder and the ocular symptoms can be clearly established. In an indirect way, however, by interference with assimilation and nutrition, the gastric or intestinal disease may be an important factor in the production, or at least in the aggravation, of ocular lesions.

Numerous observations have been published concerning affections of the eye attributed to decaying or decayed teeth. Among these are to be found cases of inflammation of the conjunctiva (of the phlyctenular type), cornea,

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[1] Mooren, Hauteinflüsse und Gesichtsstörungen, 1884.

[2] Foerster, Graefe-Saemisch, Handbuch der gesammten Augenheilkunde, Bd. vii.

[3] Nieden, Centralblatt für praktische Augenheilkunde, December, 1887.

[4] Rothmund, Archiv für Ophthalmologie, Bd. xiv. Abth. 1.

[5] The children of two of the families were related (cousins), and eleven of the fourteen belonged to these two families. In the third family the children (three) were the offspring of cousins.

and iris. Amblyopia, of varying degree, without ophthalmoscopic changes, has also been recorded by several writers.[1] Optic neuritis occurring in patients suffering from dental disease, of which numerous examples have been recorded, is usually secondary to orbital periostitis or cellulitis following suppuration in the antrum.

Perhaps the most frequent ocular disturbance noted in connection with dental irritation is that of accommodative weakness. Schmidt,[2] especially, has drawn attention to this association, and has published a series of observations. Out of ninety-two cases, he found limitation of accommodation in seventy-three; the condition was most frequent in young individuals. The reverse of this—namely, spasm of accommodation—has also been noted in cases of disease of the teeth.

Much more serious than a functional disturbance, such as failure of accommodation, is the optic nerve atrophy which is an occasional sequel of hemorrhage from the gastro-intestinal tract. As is well known, loss of, or great defect of, sight may be the immediate result of severe hemorrhage from any source, and whether spontaneous or traumatic. It is, however, much more common after spontaneous hemorrhage than after bleeding from accidental or operative wounds,[3] and, according to most authorities, bleeding from the gastric mucous membrane is the most frequent cause. Fries[4] found that in a series of ninety-six cases the hemorrhage was from the gastro-intestinal tract in thirty-five per cent.

The visual defect in these cases may vary from a slight degree of amblyopia to complete blindness, and may be temporary or permanent. Leber states that the loss of vision is usually greater when the bleeding is from the stomach than when it is from the bowels, and Fries's investigations tend to support this statement and show that improvement in sight is more common after intestinal than after gastric hemorrhage. According to Gowers, *complete* restoration of sight has never been recorded in a case of blindness following hemorrhage from the stomach. Indeed, it appears established that even partial recovery is less probable in cases of hæmatemesis than in any other form of spontaneous bleeding. The loss of sight, which is sudden or rapid, may be noted at the time of, or immediately after, the hemorrhage; the patient, on recovery from the syncope induced by the loss of blood, is found to be blind. In other instances it occurs shortly after,—*i.e.*, within a few hours of the attack, while in the majority of cases two to five days elapse between the hemorrhage and the onset of the amblyopia. The interval may, it is stated, be as much as three weeks (Berger).

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[1] *Vide* Hutchinson, Royal London Ophthalmic Hospital Reports, vol. iv. p. 381; Gill, Nagel's Jahresbericht, 1872; Samelsohn, Ibid., 1877; Bull, International Dental Journal, March, 1898.

[2] Schmidt, Archiv für Ophthalmologie, Bd. xiv. Abth. 1, S. 107.

[3] Fries, in an analysis of one hundred and six cases, found only five in which the hemorrhage was traumatic in origin.

[4] Fries, Klinische Monatsblätter für Augenheilkunde, August, 1876.

Both eyes are affected, as a rule, and inequality in the degree is, according to Fries, uncommon. In about ten per cent. (of cases following hemorrhage from all sources) the affection is unilateral.

When ophthalmoscopic examination has been made soon after the onset of symptoms, changes in the retinæ and papillæ have usually been found. These vary from appearances of slight retinal œdema to signs indicative of acute neuro-retinitis with hemorrhages and white patches (Foerster, Schweigger, Hirschberg, Landesberg, and others). In a few of the cases examined at a later period, little or no change in the fundus has been found, but in a large majority the optic disks are atrophied, the retinal vessels much shrunken, and there are signs of degeneration of the retina.

This serious ocular complication of gastro-intestinal disorders is most common in cases of severe hæmatemesis in patients suffering from chronic alcoholism. It may, however, also be met with in hemorrhage due to gastric ulcer, malignant disease of the stomach or intestines, or to hemorrhoids. The following is a brief record of a case:

Mrs. A., aged forty-eight, had very severe hæmatemesis in the early morning of September 30, and became collapsed. No recurrence of the hemorrhage took place. On the morning of October 3 the sight failed rapidly. On the 5th there was partial recovery of vision, but on the 6th it again sank to complete blindness, which was permanent. She was seen by the writer on October 14, when the condition was briefly this: Pupils motionless to light; no perception of light. Ophthalmoscopic examination revealed, in each eye, a very pale, hazy, slightly œdematous papilla, surrounded by cloudy retina, by which the veins, which looked turgid, were partially obscured. The arteries were narrowed; no hemorrhages were seen. The patient had a slightly enlarged liver, and deforming arthritis of her hands; she was much addicted to alcohol. There was no albumen in the urine. She was not seen again by the writer, but was reported by her medical attendant to have remained quite blind.

No wholly satisfactory explanation of these cases has been given; von Graefe suggested retro-ocular hemorrhage as a cause, and others have attributed the symptoms to inflammation of the optic nerves, to a disturbance of the circulation in the optic nerve and retina, and to rapid degeneration of the retinal vessels induced by the acute anæmia. Gowers[1] expresses the opinion that "one effect of the loss of blood may be upon the retinal elements themselves." It is to be borne in mind that cases of blindness following hemorrhage from the gastro-intestinal tract always occur in individuals who are suffering from disease of these organs. So that, in addition to the loss of blood, which is not necessarily excessive, there may be other factors concerned in the production of the optic nerve lesions.[2]

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[1] Medical Ophthalmoscopy, 3d ed.

[2] Further information concerning the effect of severe hemorrhage upon the optic nerves will be found in the article on Ocular Lesions in Diseases of the Circulatory System.

In some of the less serious gastro-intestinal disorders, ocular symptoms may occur. The recorded observations are not very numerous, and the connection, if any, between the abdominal and the ocular conditions is generally uncertain. Atrophy of the optic nerve has been noted by Galezowski[1] in association with chronic gastric disorders; and Immermann[2] has recorded a case of blindness from optic atrophy following a severe attack of diarrhœa. Immermann's case was a remarkable one in a boy aged fourteen and a half, in whom excessive purging followed the administration of calomel and jalap. During the succeeding night the boy became completely blind, and at a later stage the optic disks showed well-marked atrophic changes. The case was one of incipient typhoid fever. The liability to chronic glaucoma induced by persistent diarrhœa (Foerster), and to acute glaucoma by constipation (Wicherkiewicz), should be attributed not to the intestinal disorder, but to the general nutritive and circulatory disturbance engendered thereby.

Eales,[3] some years ago, described a group of cases of recurrent hemorrhage in the retina, and into the vitreous from the retinal vessels, associated with epistaxis, in young males in whom chronic constipation and high arterial tension were present. Eales held that constipation was the main factor in the production of the retinal hemorrhage in these individuals. In none of his patients was there evidence of retinitis, or of any constitutional disease,—*e.g.*, syphilis, leucocythæmia, etc. Hutchinson[4] has also written on this class of cases, but he attaches less importance to constipation as a cause of the intra-ocular hemorrhage. These cases, albeit rare, are now fairly well known, and although the association of symptoms noted by Eales has been repeatedly observed, it seems most probable that there are more factors than one concerned in the production of the ocular lesions, and that constipation in young people does not *per se* lead to extravasation of blood from the retinal vessels.

In disease of the liver, if accompanied by jaundice, the subjective symptom of yellow vision (xanthopsia) is occasionally noted. This phenomenon is present in a very small percentage of cases; Hirschberg reported five in one thousand. It is most frequent in catarrhal jaundice, and is usually noted as an early symptom. The explanation formerly given, and generally accepted, that the sensation was due to a yellow tint of the refractive media has been called in question, and it is suggested that the symptom may be the expression of a toxic effect upon the retina. It is almost certainly a peripheral lesion, and in some of its features is analogous to the yellow vision of santonin-poisoning (Knies).

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1 Galezowski, L'Union médica'e, 1876.

2 Immermann, Archiv für Psychiatrie und Nervenkrankheiten, xix. 1, 286.

3 Eales, On Cases of Retinal Hemorrhage associated with Epistaxis and Constipation, Birmingham Medical Review, 1880.

4 Hutchinson, Transactions of the Ophthalmological Society of the United Kingdom, vol. i. p. 26.

The altered condition of the blood which is present in jaundice, from whatever cause, may lead to retinal hemorrhage. Litten[1] especially has drawn attention to the (according to his investigations) frequent occurrence of blood extravasation in the retina in almost every variety of hepatic disease in which jaundice is present. Junge[2] and Stricker[3] have also recorded observations upon the occurrence of retinal hemorrhage in hepatic jaundice.

Landolt,[4] a few years ago, published some observations upon two cases in which disease of the liver, of the cirrhotic type, and retinitis pigmentosa coexisted. He considered that the hepatic and the retinal disease were closely connected, and that the structural changes in the liver and in the retina were in many ways analogous. More recently Hori[5] has reported the case of a man, aged forty-nine, who died of cirrhosis of the liver; he was greatly emaciated and jaundiced. There were ophthalmoscopic signs of disease of the choroidal vessels, but no obvious changes in the retina; the visual fields were contracted and the man was night-blind. Post-mortem examination revealed signs of a chronic inflammation of the uveal tract.

Further evidence is necessary before Landolt's views as to the relation between hepatic cirrhosis and d sease of the choroid and retina can be accepted in their entirety.

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[1] Litten, Zeitschrift für klinische Medicin, Bd. v. Heft 1.

[2] Junge, Würzburger Verhandlungen, Bd. ix. S. 219.

[3] Stricker, Berliner klinische Wochenschrift, 1874.

[4] Landolt, Archiv für Ophthalmologie, Bd. xviii. Heft 1.

[5] Hori, Archives of Ophthalmology, vol. xxvii., No. 6.

# OCULAR LESIONS IN VARIOLA, RUBEOLA MORBILLI, SCARLATINA, ERYSIPELAS, AND DIPHTHERITIS.

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## VARIOLA.

EYE-AFFECTIONS both during and after variola are extremely common, and almost every portion of the whole visual apparatus may be attacked. The most important ocular complication is inflammation and ulceration of the cornea, and it is to this cause that most of the blindness after small-pox is to be attributed. Before the introduction of vaccination, small-pox was one of the most frequent causes of blindness. It has been calculated that prior to Jenner's discovery, both in France and in Germany, thirty-five per cent. of all blind persons had lost their sight from this cause. The percentage is not nearly so high since vaccination has been introduced. Fuchs[1] states that in France the percentage amounts to seven, and in Germany ranges from two per cent. to three and six-tenths per cent.

The frequency of eye-complications in variola varies from one per cent. to eleven per cent. in the statistics published by different observers,[2] and a considerable proportion of the eyes affected are permanently blinded, usually from corneal ulceration.

The conjunctiva is commonly affected in the early stage, as in other febrile diseases, being usually congested, and occasionally presenting a catarrhal inflammation. The intensity of the ophthalmia is directly related to that of the pustular eruption in general, and more particularly to that of the eruption on the face and eyelids. If pustules form on the conjunctiva itself, the inflammation is always extremely severe, with profuse secretion and chemosis. The conjunctivitis in these cases strongly resembles that of gonorrhœal infection, but rarely proves quite so serious in its effects upon the cornea. It yields generally to treatment by mild antiseptic lotions and ointments. The hemorrhagic form of variola is nearly always marked by subconjunctival ecchymosis, which may produce intense chemosis.

The skin of the lids is commonly a site of the pustular eruption (accord-

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[1] The Causes and Prevention of Blindness, Wiesbaden, 1885.

[2] Fuchs, loco citato.

ing to one authority, in twenty per cent. of the cases), and so much swelling of the tissues may occur that the eyes cannot be opened for several days. In these cases conjunctivitis is usually severe, and may lead to corneal ulceration. The edges of the lids, too, may become ulcerated, and afterwards distorted by the subsequent cicatrization, so that misplaced cilia and occluded Meibomian ducts, hordeoli, blepharitis ciliaris, ectropium, trichiasis, distichiasis, ankyloblepharon, etc., may result. Abscesses may form also on the lids as in other portions of the skin after the subsidence of the eruption, and produce various malpositions which may require operative treatment, and may even lead to blindness from corneal ulceration at a period of time long after the small-pox has been cured.

As rare complications, periostitis and caries of the orbital border have been observed.

Small-pox pustules occasionally form upon the conjunctiva bulbi, and more rarely upon the conjunctiva palpebrarum, the plica semilunaris, and the caruncula; but never, it is said, in the fornix. Their favorite situation is on the limbus, where they frequently produce corneal ulceration. They do not form upon the cornea itself, according to most modern authorities.[1] The pustules on the conjunctiva are usually much smaller than those which form on the skin, and in their appearance and course resemble greatly the pustular inflammation observed in strumous children.

Although the cornea, according to modern observers, is not the site of actual pustules, it becomes frequently ulcerated, either in consequence of conjunctival affections or more usually quite independently.

The corneal complications may arise in various ways. They may be caused by the primary conjunctivitis without any pustules on the conjunctiva, but more frequently by actual pustules on the conjunctiva, especially when situated on the limbus. They may arise out of a secondary conjunctivitis produced by the contagium transmitted from broken-down pustules on the skin of the eyelids or elsewhere, or they may originate without any ophthalmia, which is the type of corneal complication most frequently observed, and is to be regarded as an endogenous inflammation due to germs circulating in the blood. From what is stated above it is manifest that the care of the eyelids and conjunctiva is an important branch of the treatment of a case of small-pox, and antiseptic lotions and ointments should be used continually even in the mildest attacks of the disease.

The serious forms of keratitis do not commonly present themselves until the fourth stage (stadium exsiccationis), and exhibit every gradation from superficial circumscribed inflammation to deep infiltrations, abscesses, and hypopyon, ending in perforation, with prolapse of iris and occasionally suppurative panophthalmitis. Extensive corneal suppuration is often accompanied by severe complications in other organs, and a fatal termination is not uncommon after both eyes have been lost.

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[1] Förster, Graefe und Saemisch's Handbuch der gesammten Augenheilkunde, Bd. vii.

This metastatic keratitis is the cause of the vast majority of cases of post-variolous blindness.

Keratomalacia may occur in variola as in other exhausting diseases as a forerunner of fatal collapse. It has been observed as early as the eighth day.

Geissler[1] has observed the permanent disappearance of a corneal opacity during an attack of small-pox which destroyed the other eye.

The uveal tract is not so frequently affected in variola as the cornea, although iritis, chorioiditis, and cyclitis occur after small-pox as they do after febris recurrens. It has been calculated that uveal affections form fourteen per cent. and corneal affections thirty-five per cent. of the eye-complications of the disease, and one observer found iritis as a primary disease in nine cases out of fifty-eight post-variolous eye-affections.

Uveal inflammations also do not commonly appear before the fourth stage (stadium exsiccationis). The iritis is usually of the so-called serous type, and is commonly associated with chorioiditis and opacities in the vitreous. It occurs most frequently after the exanthem has disappeared, in the period when abscesses and boils begin to develop. In consequence of the lateness of its occurrence, it is described by Hirschberg as "post-variolous" iritis.

Circumscribed chorioidal inflammation is rare. It is recorded by Knies.[2] Suppurative chorioiditis of metastatic origin also occurs, according to Fuchs. The so-called ciliary irritation, which is found sometimes as early as the fifth day, is often only a symptom of corneal or iritic disease, but may exist independently. It is characterized by slight pericorneal injection, photophobia, lacrymation, tenderness on pressure, and small motionless pupil. It may persist for a considerable time after the termination of an attack of small-pox, and appears to be more frequent in the milder than in the severe cases of the exanthem.

As the result of chorioidal inflammation with implication of the vitreous humour, posterior polar cataract occasionally develops. Glaucoma also has been observed by Adler during variola,[4] and similar cases are recorded by v. Graefe, Watson, and Coccius. Manz has recorded two cases of retinitis after small-pox,[5] and Adler three of neuro-retinitis diffusa and nephritica which appeared in the stage of desiccation.[6] Uræmic amaurosis in variola is rare, according to Leber,[7] although renal disease is not so infrequent as a complication or sequela of small-pox, and albuminuria may produce eye-lesions.

[1] Jahresbericht der Ophthalmologie von Nagel, 1872, S. 266.

[2] Die Beziehungen des Sehorgans und seiner Erkrankungen zu den übrigen Krankheiten des Körpers und seiner Organe, Wiesbaden, 1893.

[3] Text-Book of Ophthalmology, translated by Duane, 1893.

[4] Jahresbericht der Ophthalmologie von Nagel, 1874, S. 378.

[5] Ibidem, 1871, S. 180.

[6] Ibidem, 1874, S. 416.

[7] Graefe und Saemisch's Handbuch der gesammten Augenheilkunde, Bd. v.

Hemorrhage may occur in the optic nerve, Knies[1] attributing to this cause some of the neuritis which has been observed after small-pox. Chronic dacryocystitis is a not infrequent sequel of variola, and acute dacryocystitis may also occur in the later stages of the actual disease.

*Vaccination.*—Thanks to the discovery of Jenner, small-pox is not now the scourge it was in former times, and it is only rarely that the ophthalmic surgeon of the present day has to deal with the disease or the ocular lesions resulting from it. It is, however, not to be forgotten that vaccination itself has caused ocular troubles of no insignificant character.

Phlyctenular conjunctivitis and eczematous inflammation of the skin of the face and eyelids may occur after vaccination, and are very probably caused by it. These lesions are not of great importance; but if the lids or conjunctiva are accidentally vaccinated, serious consequences may ensue. Vaccine vesicles may form on the lids without causing much trouble; but cases are on record in which malpositions of the eyelids have been produced by cicatrization of the resulting ulcers. Thompson[2] has observed ankyloblepharon in a man whose eyelid had been infected by the vaccine vesicle on his child's arm. A similar case of almost complete ankyloblepharon (as yet unpublished) was recently operated on by Arthur Benson in St. Mark's Ophthalmic Hospital, Dublin. The patient was a child who had been sleeping in the bed with a brother who had been vaccinated. After the operation the cornea was found to be normal.

The disease is generally situated at the angle of the lids, the infection being probably rubbed on in that locality by the patient's finger. After some four days itching and pain are felt, and an inflammatory œdema appears, which almost completely closes the lids. A superficial ulcer then forms, which simulates to some extent a primary syphilitic sore. The inflammatory œdema may spread over the nose and cheeks and down to the neck. The preauricular gland is swollen and tender. The conjunctiva becomes chemosed, but the cornea usually remains clear during this period. Several fresh ulcers may form along the margin of the lids. After eight to twelve days the acute stage begins to subside, and cicatrization is completed in about three weeks. Just as in variola, however, after the acute inflammation is over, corneal complications may appear in the form of interstitial keratitis, superficial or deep, accompanied by iritic congestion and deposits on Descemet's membrane. A peculiar concentric arrangement of the corneal opacity has been described by Schirmer,[3] which he regards as possibly caused by the shrinking of a fibrinous exudation on the posterior surface of the cornea. These opacities did not clear entirely in any of Schirmer's cases, though they became less dense. Most of the recorded instances recovered with but trifling permanent opacity, but in others sup-

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[1] Loco citato.

[2] Transactions of the Ophthalmological Society of the United Kingdom, vol. xii. p. 82.

[3] Bericht über der Ophthalmologischen Gesellschaft zu Heidelberg, 1891, S. 14.

puration appeared and dense opacities resulted. Hypopyon and perforation of the cornea are possible. I was consulted in 1894 by a medical man, whose case is worth recording for its severity, for the accuracy with which the period of incubation can be determined, and also for the probability of the corneal affection being primary in this case. The infection occurred in breaking a capillary tube of lymph, when something went into his left eye, whether glass or fluid could not be determined. He washed the eye, and forgot all about the occurrence for four days. His eye then began to become itchy and painful and inflamed. I saw him fifteen days after inoculation, and found general conjunctival vascularity, with a gelatinous haze of the whole cornea and vascularity of its upper border. Three weeks later I again saw him, and found the cornea clear, except for a curved, infiltrated, almost marginal, ulcer above, with dense pannus at the corresponding edge and a faint infiltration more centrally. A small hypopyon was present. Under treatment by cauterization and antiseptics the ulcer gradually healed and the hypopyon absorbed, returning twice, however, before final absorption. A small ulcer remained after the hypopyon had finally disappeared, which ulcer perforated one day without warning or apparent reason, and then promptly healed, leaving a dense leucoma without iritic adhesion. At no time were any of the concentric rings of opacity described by Schirmer present.

Pemphigus of the conjunctiva has been observed after vaccination by Tilly.[1] The condition resulted in blindness of both eyes. Suppurative chorioiditis of metastatic origin may also follow vaccination, as it does variola and other acute infectious diseases.[2]

## VARICELLA.

Acute iritis with hypopyon has been observed after varicella, and one observer has recorded a case in which the eruption on the eyelids preceded that on the rest of the skin.

## RUBEOLA MORBILLI.

Ocular lesions present themselves at the commencement of an attack of measles, and are usually among the earliest symptoms which attract attention even before the eruption of the exanthem upon the skin. After the febrile attack has subsided, ocular troubles, which doubtless depend upon the effects of the dyscrasia, are also frequently experienced. These may persist for a long period of time after the actual attack of measles has passed off. In addition to these common complications, a certain number of rare cases of serious ocular lesions occurring during or shortly after an attack of measles have been recorded.

The onset is usually accompanied by well-marked conjunctival catarrh,

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[1] Ophthalmological Report of the American Medical Association, Ophthalmic Review, vol. vi. p. 241.

[2] Fuchs, loco citato.

which presents the ordinary signs and symptoms of that disease. The mucous membrane is congested, its secretion increased, and photophobia and lacrymation are present, the former sometimes to an extreme degree. Corneal complications are extremely rare, and the conjunctival affection usually passes off, even without treatment, before the stage of desquamation is reached. For treatment it is usually sufficient to wash the conjunctiva with solution of boric acid, and to apply a little vaseline to the edges of the lids at night. If the photophobia is excessive, it can generally be relieved by instilling a solution of sulphate of atropine or hydrochlorate of cocaine, or a combination of the two, into the conjunctival sac.

More vigorous treatment, such as nitrate of silver solution, etc., is rarely required, but occasionally after the acute attack passes off a chronic conjunctivitis remains, which is to be treated in the ordinary way. Förster[1] states that eyes are in exceptional cases destroyed by diphtheria, ulcerative keratitis, or keratomalacia immediately after an attack of measles. Fuchs[2] alludes to the occasional occurrence of a diphtheritic or blennorrhœal conjunctivitis in the early stage, which, however, is not to be confounded with true blennorrhœa or diphtheria. In these cases the cornea is endangered. Fuchs has also observed inflammation and suppuration of the Meibomian glands in the stage of convalescence.

The skin of the lids is of course just as liable to be affected by the exanthem as the skin elsewhere. After the general disease has subsided, a number of strumous eye-affections often show themselves during the succeeding months. The principal of these are catarrhal and phlyctenular ophthalmia, superficial vascular keratitis, and marginal blepharitis. They are amenable to the usual local and general treatment of such affections.

Among other troubles during this period, paresis of accommodation may occur.

Lesions of the deeper parts of the visual apparatus have been occasionally seen during an attack of measles, or after the attack has passed off. Nagel[3] has recorded the case of a boy, aged eight years, who on the fourth day became comatose, and developed convulsions with opisthotonos and dilated pupils. The coma disappeared in ten days, and the eyes were then found to be blind, the pupils reacting, however, sluggishly to light. There were no ophthalmoscopic lesions visible, and the sight improved during the next three or four weeks. Speech and locomotion were also affected. Finally, the vision became nearly completely restored under treatment by strychnine hypodermically.

Nagel notes in the same publication two cases of bilateral and perma-

[1] Förster, Graefe und Saemisch's Handbuch der gesammten Augenheilkunde, Bd. vii. S. 161.

[2] Fuchs, E., Text-Book of Ophthalmology (English edition), 1893, p. 97.

[3] Nagel, Behandlung der Amaurosen und Amblyopien durch Strychnin, Tübingen, Jahresbericht der Ophthalmologie von Nagel, 1871, S. 348.

nent blindness after measles, in one of which optic neuritis was present. The epidemic in which Nagel's cases occurred was characterized by severe head-complications and deaths from meningitis, which is rare in measles.

A. v. Graefe[1] has recorded a case of bilateral blindness during convalescence from measles. The ophthalmoscope revealed a delicately diffused haziness of the papilla and neighboring retina, which rapidly cleared off. Normal sight was obtained in eight weeks.

Leber[2] states that he has been unable to find any undoubted case of uræmic amaurosis in measles, and that only one case of albuminuric retinitis after measles was then on record,—viz., one published by Horner, in *Klinische Monatsblätter für Augenheilkunde* for 1863.

Carreras Arago[3] has observed optic neuritis in a case of measles in which meningitis was present. This case terminated fatally. In another patient the same observer has seen extreme amblyopia with contracted retinal arteries.

Boucher[4] records the case of a man whose sight became affected some three weeks after an attack of measles. The loss of sight was preceded by a deep semi-comatose sleep, and accompanied by some uncertainty and feebleness in muscular movements. The fundus oculi was normal at first, but some two weeks later there was a delicate grayish haziness of the retina and papilla. Partial atrophy of the nerves resulted. Boucher attributes the blindness to a lesion of the occipital lobes, basing his conclusions principally upon the persistence of the pupillary reflex, and upon the fact that the ophthalmoscopic changes were not present till two weeks after the blindness. He quotes a case of Calmeil's, in which blindness following measles was considered to be due to affection of the occipital lobes, the post-mortem, however, being made several years after the blindness appeared.

Woods[5] records two cases of blindness after measles, due to double optic neuritis, which apparently came on during the stage of convalescence. He divides the cases of blindness after measles into two classes: (1) those showing no eye-lesions till late in the history of the case; (2) those with marked neuritis from the beginning. The former are apparently due to cerebral lesion, probably vascular, with consecutive nerve-disease; the latter, to basal meningitis and neuritis.

The evidence, however, does not seem conclusive that the cases of

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[1] V. Graefe, Ueber Neuroretinitis und gewisse Fälle fulminirender Erblindung, Archiv für Ophthalmologie, Bd. xii. 2, S. 138.

[2] Leber, Th., Graefe und Saemisch's Handbuch der gesammten Augenheilkunde, Bd. v. Ss. 811, 955, 968.

[3] Carreras Arago, Die Masern und ihre verschiedenen primitiven und consecutiven Manifestationen am Auge, Centralblatt für praktische Augenheilkunde, October, 1882.

[4] Boucher, Rougeole Amblyopie d'Origine cérébrale, Société d'Ophtalmologie de Paris, Recueil d'Ophthalmologie, 1888, p. 716.

[5] Woods, H., Optic Neuritis after Measles and Intermittent Fever, Archives of Ophthalmology, vol. xxi. p. 95.

optic atrophy supposed to be instances of central lesion may not be due to retrobulbar neuritis of basal origin, as was held by Albrecht von Graefe.[1] The delicate grayish haziness of the retina and papilla which has been observed in these cases is not easily accounted for if the primary lesion is restricted to the visual centres.

Wadsworth[2] records three instances of optic neuritis after measles, all with cerebral symptoms, and one with paralysis of the abducens. The last case recovered; of the other two, one died of meningitis, and the second recovered with blindness from atrophy of the optic nerves.

Optic neuritis may also occur as a secondary result of measles, the connecting link being furnished by otitis media.[3] The ocular lesion is either the result of meningitis due to the ear-disease, or is one of the effects of general pyæmia.

Fieuzal[4] describes spontaneous gangrene of the eyelid as a sequel of measles, and Lindner[5] attributes dacryoadenitis to the same cause.

Finally, measles is not an unmixed evil, for it has had the happy effect of curing pre-existing trachoma and pannus in a case recorded by Fialkowsky.[6]

## SCARLATINA.

The principal ocular lesions which have been observed in this affection are catarrhal conjunctivitis, which occurs in this as in other acute exanthemata, and amaurosis or amblyopia, which is generally the result of renal complications. The skin of the eyelids is, like the rest of the skin, affected by the exanthem in many cases, but not to such an extent as to be of any importance. The conjunctivitis needs no special description, as it differs in no important respect from that occurring in measles.

Many cases of amaurosis or amblyopia are on record. These are either complications during an attack of scarlatina, or occur shortly after the subsidence of the fever.

Ebert[7] has recorded three cases of sudden blindness coming on after convulsions and stupor. All the cases recovered. In one, ophthalmoscopic examination gave a negative result. The pupils reacted to light,

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[1] Loco citato.

[2] Wadsworth, Neuritis after Measles, Transactions of the American Opththalmological Society, July, 1880; Archives of Ophthalmology, ix. 3, p. 341.

[3] Keller, Neuritis optica bei Mittelohrerkrankung, Monatsschrift für Ohrenheilkunde, June, 1888, S. 149.

[4] Fieuzal, Sphacèle de la Paupière suite de Rougeole, Jahresbericht, 1887, S. 422; Centralblatt für praktische Augenheilkunde, 1887, S. 484.

[5] Lindner, L., Ueber die Behandlung einiger Augenerkrankungen nach erlöschener Influenza Epidemie, Wiener Medicinische Wochenschrift, Nos. 16 and 17; Jahresbericht, 1891, S. 448.

[6] Fialkowsky, Ein Fall von Heilung mehrerer Augenerkrankungen unter dem Einfluss von Masern, Sitzungsbericht der Medicinische Gesellschaft zu Dünaburg; Jahresbericht, 1888, S. 538. Trachoma and Pannus cured by an Attack of Measles.

[7] Förster, Graefe und Saemisch's Handbuch der gesammten Augenheilkunde, Bd. vii. S. 162.

which enabled him to locate the lesion above the corpora quadrigemina, between these bodies and the visual centres. Albuminuria was present in all the cases.

Förster[1] cites a case of blindness lasting for sixteen days. In this case there was albuminuria, but there were no convulsions.

Monod[2] has observed absolute amaurosis ushered in by convulsions and lasting for four days. Albuminuria was present, and the pupils reacted to light.

Power[3] has described a case in which albuminuria had existed for ten days before a sudden and complete amaurosis. The latter lasted for five days, and vision was then gradually restored in three weeks.

Loeb[4] records a case of uræmic convulsions and dropsy, with amaurosis and paresis of the left arm. The irides reacted to light, and vision returned in twenty-four hours. The motor lesion remained. He cites another case in which the amaurosis preceded the convulsions.

Martin[5] has observed sudden and temporary amaurosis in scarlatinal nephritis.

The above cases are quoted by Förster,[6] who calls attention to the fact that in all of them albuminuria was present, and that the amaurosis occurred in the desquamation stage after a period of generally favorable symptoms. The amaurosis was ushered in by cerebral symptoms, headache, convulsions, vomiting, and stupor. It came on suddenly, was bilateral, and for a time was complete. No ophthalmoscopic lesions were detected, and the blindness gradually cleared off. There can be no doubt that these cases must be classed as uræmic.

A. v. Graefe at one time advocated the theory that the amaurosis above described is differentiated from uræmic amaurosis by the reaction of the pupils; but cases of undoubted uræmic amaurosis with no connection whatever with scarlatina have been observed with the same pupillary phenomena, so that this argument against the uræmic origin of the amaurosis cannot hold good.

Retinitis albuminurica occurs after scarlatina, but is not frequent. It is

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[1] Förster, R., Scharlach mit nachfolgender Nierenerkrankung und transitorische Erblindung, from Jahrbuch für Kinderheilkunde und physische Erziehung in Klinische Monatsblätter für praktische Augenheilkunde, 1872, S. 346.

[2] Monod, Albuminurie aiguë consécutive à la scarlatine, convulsions épileptiformes, amaurose, guérison, Gazette des Hôpitaux, 1870, p. 113; Jahresbericht der Ophthalmologie von Nagel, 1870, S. 370.

[3] Power, Case of Complete but Temporary Loss of Vision in an Attack of Scarlet Fever, Practitioner, May, p. 257; Jahresbericht der Ophthalmologie von Nagel, 1871, S. 341.

[4] Loeb, Transitorische Erblindung und persistirende Paralyse nach Scharlach, Jahrbuch für Kinderheilkunde, Bd. viii. S. 194; Förster, loco citato.

[5] Martin, St. Bartholomew's Hospital Reports, 1867, p. 246. Temporary Blindness after Scarlatinal Nephritis with Dropsy, Förster, loco citato.

[6] Förster, Graefe und Saemisch, Handbuch, loco citato.

more a complication of the chronic form of Bright's disease than of the croupous nephritis which is found in scarlatina. The prognosis is more favorable than in albuminuric retinitis unconnected with an exanthem, but partial optic atrophy has been observed.[1] The sight can also be affected as the result of scarlatina without the presence of albuminuria or any evidence of kidney-disease, as in the case recorded by Hodges,[2] where thrombosis of the central artery was observed in one eye of a growing girl while recovering from scarlatina. In this case the urine was normal, so that the ocular lesion may possibly be attributed to debility induced by the disease.

Pflüger[3] has observed papillo-retinitis after scarlatina without kidney-affection, and the same phenomenon has been observed by Betke,[4] though in the latter case the patient had suffered at an earlier period from hemiplegia. Leber[5] records a case of a boy who became blind without ophthalmoscopic signs and with normal urine. The only assignable cause was latent scarlatina, and Leber seems to regard this peculiar case as somewhat analogous to the post-diphtheritic lesions in other nerves.

Occasionally accommodative asthenopia shows itself after scarlatina as it does after measles. It may persist for a very long period of time even after the general health is completely re-established.

Scarlatina may indirectly be the cause of serious ocular lesions. Förster[6] instances the case of a boy of nine years who suffered from caries of both temporal bones and complete paralysis of both facial nerves. Both corneæ were destroyed in consequence of the lagophthalmos, and the unfortunate patient became blind as well as deaf. Fuchs, in his Text-Book, states that suppurative chorioiditis of metastatic origin may occur in scarlatina as in typhus, variola, etc. In most if not in all of the recorded cases suppurative otitis has preceded the chorioiditis (or retinitis).

Phillips[7] has observed an œdema of the upper eyelid during scarlatina which also is apparently associated with suppurative otitis. The swelling was not white and doughy as in renal dropsy, but tense and livid, and in the cases recorded it was most marked before rupture of the membrana tympani, and increased afterwards if the discharge from the ear became less free. Phillips considers it probable that the affection is connected with thrombosis of the cavernous sinus.

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[1] Horner, Zur Retinalerkrankung bei Morbus Brightii, Klinische Monatsblätter für Augenheilkunde, 1863, S. 71; Höring, Retinalerkrankung bei Morbus Brightii, ibidem S. 215.

[2] Ophthalmic Review, vol. iv. p. 296.

[3] Pflüger, Neuritis Optica, Archiv für Ophthalmologie, Bd. xxiv. 2, S. 180.

[4] Betke, Amblyopie nach Scarlatina, Klinische Monatsblätter für Augenheilkunde, 1869, S. 201.

[5] Leber, Graefe und Saemisch's Handbuch der gesammten Augenheilkunde, Bd. v. S. 1048.

[6] Loco citato.

[7] Phillips, Sydney, Cases of Œdema of the Upper Eyelid during Scarlet Fever, British Medical Journal, 1895, vol. i. p. 194.

## ERYSIPELAS.

Erysipelas of the face may affect the eyes in various ways. It rarely fails to implicate the skin of the eyelids, and may, by the formation of abscesses, produce such defects of position as ectropium and lagophthalmos, that may be the means of setting up corneal ulceration, and so lead to blindness long after the erysipelas has itself passed away. The orbital tissues may be affected. If so, the condition is usually manifested by the occurrence of exophthalmos. Pus may form in the orbit, in the lacrymal sac, or in the eyeball itself, and the globe be destroyed by purulent ophthalmitis. In other cases permanent defects of motion of the globe may be caused by the inflammatory processes in the orbit, the function of muscles being abolished, or cicatricial adhesions binding the eyeball to some portion of the orbital contents or the orbital walls. The cornea may be primarily affected, as in a case recorded by Cousserant, where bullous keratitis occurred in erysipelas complicated with albuminuria,[1] and cyclitis has been observed in a case published by Cornwell, who attributed the intra-ocular lesion to septic embolism.[2] Finally, the disease may spread to the cranial cavity, and death may occur from purulent meningitis.

From an ophthalmological stand-point, however, the most interesting complication of erysipelas is atrophy of the retina and optic nerve, which follows the facial type of the disease, and is the usual cause of post-erysipelatous blindness. It comes on in most cases with remarkable rapidity. The sight may be perfect at the time the erysipelas is first observed on the face and eyelids, and the eye may be found completely amaurotic as soon as the swelling has subsided sufficiently to enable the lids to be again opened. In all the observed cases the extreme contraction of the retinal vessels, especially of the arteries, has been the most prominent ophthalmoscopic appearance.

In the cases described by Jäger[3] the atrophy of the vessels was much more marked than in the ordinary cases of white atrophy of the optic nerve, and in those recorded by Pagenstecher[4] the diminution of size in the vessels, more especially the arteries, was very striking, although in his second case vision was not completely destroyed. The affection was in this case bilateral, with central scotoma and no contraction of the peripheral fields of vision.

Förster[5] attributes the retinal lesion in these cases to a direct transmission of the inflammation along the sheaths of the vessels rather than to a compression of the optic nerve exercised through the medium of the orbital tissues, and this view is strongly supported by the evidence afforded in

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[1] Jahresbericht der Ophthalmologie von Nagel, 1876, S. 7.
[2] Ibidem, 1882, S. 380.
[3] Ophthalmoscopischer Hand-Atlas, Taf. x. Fig. 51, und Taf. xvi. Fig. 75.
[4] Klinische Monatsblätter für Augenheilkunde, Bd. viii. S. 207.
[5] Graefe und Saemisch's Handbuch der gesammten Augenheilkunde, Bd. vii. S. 153.

all recorded cases, although the direct implication of the orbital tissues has been observed frequently, and Knapp has advocated a different theory in a most valuable paper upon post-erysipelatous blindness, published in 1884.[1] In it there is a systematic review of most of the cases previously recorded, and a detailed account of one case occurring in his own practice. This latter case is peculiarly interesting, inasmuch as the eyes were examined very shortly after the onset of the disease, although it is only fair to state that perhaps some uncertainty attaches to the case on account of the coexistence of tertiary syphilis. The ophthalmoscopic appearances were very remarkable. At first there was a milky-white fundus, with dark, almost black, tortuous vessels radiating from the disk, which was itself invisible. These vessels tapered off towards the disk, and were regarded as veins gorged with stagnant blood. Numerous dark red hemorrhages were scattered over the fundus. Two days later small light red arteries could be seen between the veins. Finally, white atrophy followed, and the vessels exhibited white perivascular lines and were interrupted by perfectly white segments, which Knapp characterizes as "white thrombi." Knapp's general conclusion in the paper is that the intra-ocular changes in blindness after erysipelas are the results of the compression of the central vessels caused by orbital cellulitis. The latter condition was observed in every case recorded except three, and its existence is probable in the three exceptions. The mortality in the cases collected by Knapp was very high,—ten deaths in thirty-five cases. Of the survivors, sixteen per cent. were blind in both eyes, sixty per cent. in one eye, while twelve per cent. recovered partial vision and twelve per cent. perfect vision.

Ophthalmoscopic appearances similar to those in Knapp's case, except for the absence of hemorrhages, have been observed by Carl.[2] He also attributes the retinal changes to interrupted blood-supply, but suggests that the ischæmia itself is brought about by an invasion of micrococci into the perivascular lymph-spaces. This view is essentially the same as that advocated by Förster, and harmonizes better with the ophthalmoscopic appearances in all the published cases than a simple compression-ischæmia does.

One interesting case has been recorded by Scougal,[3] which differs from the typical form of the eye-affection, and is not easily explicable by either hypothesis. The erysipelas was only one-sided, and the sight was not affected till five weeks after recovery. No evidence of orbital cellulitis was present at any time, and the onset of the blindness was gradual. Both eyes were affected, and the diminution of sight was progressive, ending in complete loss of vision of one and nearly complete blindness of the other eye.

Vossius[4] describes a case of neuro-retinitis with detachment of the retina, and retinal and subretinal exudations, which followed on several

[1] Archives of Ophthalmology, vol. xiii. p. 83.

[2] Klinische Monatsblätter für Augenheilkunde, 1884, S. 118.

[3] Transactions of the Ophthalmological Society of the United Kingdom, 1893, p. 82.

[4] Klinische Monatsblätter für Augenheilkunde, 1883, S. 294.

occasions three or four attacks of facial erysipelas. He also records another case in which neuro-retinitis was observed after an attack of erysipelas, with disproportionately extreme defect of sight in both eyes. This patient recovered with perfect vision.

Galezowski[1] attributes to erysipelas a peculiarly malign influence in glaucoma, which is easily intelligible when it occurs as a complication of iridectomy or sclerotomy, when it should lead to the loss of the eye. Apart from its influence upon operations, there does not appear to be any direct connection between erysipelas and glaucoma.

Some curious cases are on record in which an attack of erysipelas has had a beneficial effect upon some pre-existing ocular disease. Walb[2] describes a case of hyalitis and (so-called) keratitis punctata of many weeks' duration which was cured by an attack of facial erysipelas.

Cocci[3] records the beneficial effect of an attack of erysipelas upon trachoma with pannus corneæ.

Nieden[4] cites two instances of uveal affections which were each cured by an attack of facial erysipelas. In both mercurial treatment had been adopted, with unsatisfactory results. The first was a case of iritis serosa with dense deposits on Descemet's membrane. An attack of erysipelas cleared away the deposits in both eyes completely, and the remarkable phenomenon was subsequently observed that the eye-affection returned in a less severe form when the patient had fully recovered from the erysipelas. This second attack of "Descemetitis" cleared off slowly under iodide of potassium. Nieden's second case was one of chorioiditis disseminata, recent in one eye and of old standing in the other. The latter was not materially affected by the erysipelas, but the former had its vision raised within the space of one month from one-sixth to ten-fifteenths.

Considerable discussion has taken place as to the path by which erysipelas of the lids and orbit travels into the cranial cavity in those cases which have terminated fatally by meningitis.

Two theories have been advocated: one, that the inflammation travels along the lymphatics; the other, that it spreads by the blood-vessels,—viz., the veins. It is probable that the latter theory is the more nearly correct, as has been shown in the exhaustive paper of Berlin,[5] and in Leber's article on the subject.[6]

Knies[7] quotes a case of acute inflammation of the lacrymal gland, observed by Carré during an attack of erysipelas, and states his belief that this complication must be of much greater frequency than is commonly supposed.

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[1] Jahresbericht der Ophthalmologie von Nagel, 1876, S. 328.

[2] Centralblatt für praktische Augenheilkunde, June, 1877.

[3] Jahresbericht der Ophthalmologie von Nagel, 1884, S. 429.

[4] Centralblatt für praktische Augenheilkunde, March, 1885, S. 80.

[5] Graefe und Saemisch's Handbuch der gesammten Augenheilkunde, Bd. vi. S. 504.

[6] Archiv für Ophthalmologie, Bd. xxvi. 3, S. 212.

[7] Die Beziehungen des Sehorgans und seiner Erkrankungen zu den übrigen Krankheit n des Körpers und seiner Organe, Wiesbaden, 1893.

DIPHTHERITIS.

Without any conjunctivitis having been present, the eye is frequently the seat of post-diphtheritic lesions. These occur in the form of paralysis or paresis, and the favorite seat of the lesion is in the accommodative apparatus. Paralysis in various situations is not uncommon after diphtheria; the velum palati, the extremities, the trunk, the bladder, and the rectum are all liable to be affected, and the external ocular muscles do not escape. It appears, however, as if the ciliary muscles were more liable to this affection than any other portion of the muscular system, with the exception of the velum palati.

We owe our knowledge of this interesting lesion to Donders. It is only occasionally that the paralysis of accommodation is accompanied by a lesion of the sphincter iridis, although Donders seems to have observed it more frequently than others have done. Scheby-Buch[1] had only one case of wide and sluggish pupil among twenty-four of paralysis of accommodation after diphtheritis. Förster had never seen the pupil affected in any of the cases he observed up to the time of the publication of his article in Graefe und Saemisch's *Handbuch der gesammten Augenheilkunde*.[2]

Völckers calls attention to the absence of the advance of the central portion of the iris in accommodative efforts as an objective sign of the lesion.[3]

The affection is nearly always bilateral, although cases of one-sided mydriasis have been recorded,—*e.g.*, one of unilateral mydriasis with paralysis of accommodation, by Jefferson.[4]

It is possible that many unilateral cases may escape observation, and it is a fact that in the bilateral ones the two eyes are not always equally affected.

A peculiarity which has been observed in the post-diphtheritic paralysis of accommodation is a positive increase of hypermetropia,—*i.e.*, during the paralysis the hypermetropia is greater than can be rendered manifest subsequently by complete atropinization. Jacobson[5] has accounted for this by the supposition that the crystalline lens may assume a flatter curvature in long-continued paralysis of the ciliary muscle than it does in the briefer paralysis induced by atropinization. The effects of long-continued atropinization in myopic children support this explanation; but, as Förster observes, the existence of a certain range of accommodation in most of the diphtheritic cases is a fact which tells the other way. Regular astigmatism has also been observed to be present during diphtheritic cycloplegia, and has been accounted for by the theory of a dynamic lenticular astigmatism,

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[1] Archiv für Ophthalmologie, Bd. xvii. 1, S. 265.

[2] Bd. vii. S. 172.

[3] Scheby-Buch, loco citato.

[4] Medical Times and Gazette, 1873, vol. xlvii. p. 89 (vide Förster, loco citato).

[5] Archiv für Ophthalmologie, Bd. xii. S. 47.

as advanced by Dobrowolsky and Woinow, which ceases to assert itself during the presence of the paralysis.

The lesion of accommodation shows itself at various times, but usually from the third to the sixth week after the commencement of the throat-affection. It generally passes off after a few weeks, but may persist for months. Eserine appears to have some influence in shortening its duration.

The acuity of vision also may suffer, probably not from any lesion of the optic nerve or the retina, but possibly from a lenticular astigmatism due to the same cause as the abnormal hypermetropia.

The paralysis of accommodation comes on rapidly, and occasionally suddenly. It lasts for weeks or months, and gradually disappears. Most cases seem to occur in from four to six weeks after the onset of the diphtheritis or in from two to three weeks after its subsidence, and the great majority of patients have been young subjects. The lesion is observed after the mildest and most insignificant attacks as well as after the most severe. It has been stated that retinal hyperæmia is present in some instances, but the vast majority of observers have found the fundus perfectly normal.

It is not necessary to have the pharynx affected in order to induce post-diphtheritic paralysis. Diphtheritic inflammation elsewhere is equally potent. Scheby-Buch has collected cases of diphtheritic inflammation in various parts of the body which were followed by paralysis, and in which no pharyngeal diphtheritis was present.

Spasm of accommodation has also been observed after diphtheria by Adams,[1] but it did not come on till nearly a year after the throat affection.

The external ocular muscles may also be affected. The onset is stated to be extremely sudden, and the lesion rarely persists long. Ptosis has been observed occasionally, and strabismus pretty frequently. Paralysis of both interni or both externi has been recorded. The external rectus seems peculiarly liable to be affected. Remak[2] found abducens paresis in ten cases out of one hundred post-diphtheritic ocular lesions. It is possible that the phenomena in some of the cases of the latter group may be accounted for by spasm of convergence. Complete ophthalmoplegia externa has also been observed.

Lesions of sensation are infrequent, but have been recorded. Laqueur[3] observed paralysis of the sensory division of the right fifth nerve which produced neuro-paralytic keratitis. Muscular paralysis occurs after other acute diseases, such as small-pox and enteric fever; but in these cases the paralysis is rarely observed, except after severe attacks of the disease, and commonly during the early stages of convalescence. Post-diphtheritic

[1] J. E. Adams, Transactions of the Ophthalmological Society of the United Kingdom, vol. ii. p. 180.

[2] Centralblatt für praktische Augenheilkunde, 1886, S. 161.

[3] Klinische Monatsblätter für Augenheilkunde, Bd. xv. S. 228.

paralysis, on the contrary, appears after the mildest attacks, and during a state of almost perfect general health.

As regards the actual site of the lesion, the theory which is most generally accepted is that of Völckers,[1] who places the lesion in the nerve-endings in the affected muscles. It cannot be in the trunk of the oculo-motor nerve nor in the short roots of the ciliary ganglion, for the pupillary reaction to light is nearly always present and the external ocular muscles are unaffected. It cannot be in the ciliary muscle itself, for eserine can still produce its physiological effect.

The almost universally bilateral occurrence of the affection is against the supposition that the lesion is central, as is also the fact that the neighboring centres for the pupil and the ocular movements are so rarely affected. It must not be assumed, however, that a nuclear origin for this or any other paralytic lesion after diphtheria is an unreasonable hypothesis. It has not yet been demonstrated that the lesions are not nuclear, and hemorrhages have been observed post mortem in the third nerve close to its nucleus and in the nucleus itself. Förster[2] quotes the observations of Buhl and Oertel as affording some information as to the tissue-changes which may be present in the nervous system, although he throws doubts upon the diphtheritic nature of Buhl's case, the description more closely approximating that of hemorrhagic small-pox. Buhl, however, found both the anterior and the posterior roots of the spinal nerves swollen and infiltrated with blood. The blood extravasation he attributed to vascular constriction produced by an inflammatory exudation, which subsequently became absorbed or developed into connective tissue. Oertel's observations are to the same effect, and the hypothesis which is hence advanced for diphtheritic paralysis in general is that the nerves are pressed on and constricted by the connective-tissue proliferation, and recover their functions when the action of this injurious agent has ceased.

Knies[3] expresses the opinion that all these post-diphtheritic lesions cannot be accounted for by the hypothesis of hemorrhages or inflammation in the nerves or their terminations in the muscles. He holds that during diphtheria, or shortly after its subsidence, some ptomaine is produced which, among other properties, has a direct influence on accommodation. It is extremely difficult on any other hypothesis to account for the almost universally normal reaction of the pupils, the frequency of paresis rather than paralysis, and the bilateral character of the lesion.

Defects of sight not to be accounted for by paralysis of accommodation do occur after diphtheria, though rarely. Concentric contraction of the fields of vision, with defective color-vision, has been recorded by Jessop[4]

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[1] Scheby-Buch, loco citato. [2] Loco citato.

[3] Die Beziehungen des Sehorgans und seiner Erkrankungen zu den übrigen Krankheiten des Körpers und seiner Organe, Wiesbaden, 1893.

[4] Transactions of the Ophthalmological Society of the United Kingdom, vol. vi. p. 386.

and Nagel.[1] The latter records the occurrence of a distinct neuritis, an observation which has also been made by some other physicians. Whether these exceptional cases of neuritis are to be attributed to the renal complications which are so frequent in diphtheria is a question which does not as yet admit of a satisfactory answer.

Contraction of the visual field is possibly more frequent than is usually believed. According to Koenig,[2] the typical contraction of the field described by Förster as occurring in retinal anæsthesia is found in cases of post-diphtheritic paralysis of accommodation. This type of visual field is marked by the field being larger when the test-object is moved from the periphery towards the centre than when it is moved from the centre to the periphery. Koenig regards these cases as examples of anæsthesia of the retina.

Oliver[3] has described a case of double chorio-retinitis with partial degeneration of the optic nerve and lymph extravasation into the retina and the vitreous, which came on after an attack of diphtheritis. The case did not come under his observation until five years after the general disease.

Lindner[4] has recorded a case of dacryoadenitis due to diphtheria, and some cases are on record in which an abscess in the orbit has been caused by the same affection.[5]

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[1] Jahresbericht der Ophthalmologie von Nagel, 1884, S. 328.
[2] Archives of Ophthalmology, vol. xxii. 2, p. 238, transl.
[3] Transactions of the American Ophthalmological Society, 1887.
[4] Jahresbericht der Ophthalmologie von Nagel, 1891, S. 448.
[5] Knies, loco citato.

# THE OCULAR LESIONS OF INFLUENZA, DYSENTERY, CHOLERA, MALARIAL FEVER, DENGUE, AND YELLOW FEVER.

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## OCULAR MANIFESTATIONS IN INFLUENZA.

INFLUENZA, or la grippe, is an epidemic and contagious disease that is characterized by an infection, with disturbances which affect the respiratory apparatus, nervous system, and digestive tract. As the apparatus of vision is intimately connected with all the systems and organs of the economy, it necessarily becomes involved in the disturbing influence of this febrile infection; and hence it is that specialists in ophthalmology have thought that they could distinguish several periods (Gillet de Grandmont), according to the different types of the disorder (nervous, catarrhal, or gastric),—the first characterized by nervous disturbances, the second by congestive phenomena, and the third by organic failure. It has also been noticed that to each of these periods different ocular disturbances correspond. In the first it is observed that the motor muscles are painful, so that the patient prefers to keep the eyelids closed, thus avoiding muscular fatigue. In the second, or congestive period, the patients complain of phosphenes and photopsia which are always annoying, and due to phospho-margaric corpuscles produced by circulatory disturbances in the retina. When the congestion is excessive, hyalitis, with floating bodies of the vitreous, and at times miliary hemorrhages of the retina and choroid, appear. During this period chronic affections of the cornea and iris assume unexpected acute exacerbations. In the last period there appear phenomena which are calculated vividly to impress the patient. Thus, some hypermetropes, who maintain normal vision by sheer accommodative power, may suddenly lose their sight by reason of weakness of the muscle of accommodation. In the same way, others about reaching the age of presbyopia, who have had the power to hide the fact,

thanks to their power of accommodation, suddenly feel the necessity of using glasses for near work.

Some acknowledge only two forms of ocular complications in influenza, —the inflammatory and the nervous (Rampoldi). In the latter forms this author includes pains in the eyeball on the slightest movement of its muscles, ophthalmodynias, superciliary neuralgias (which may or may not be accompanied with hyperæmia of the conjunctiva or with infiltration of the cornea), paralysis of accommodation, accommodative asthenopia, and blepharospasm. In the first group he places violent hyperæmia of the conjunctiva, styes, furuncles, panophthalmitis, glaucomatous cyclitis, and hypopyon.

In a disease like influenza, where the infectious agent generalizes its effects in ways so diverse, it is not possible to adjust the ocular manifestations which accompany or follow it to a perfect classification; but it is certain that in the great majority of cases they may be placed in one or the other of the two groups above mentioned.

We cannot believe, with Cowper, of Brussels, that many of the eye-complications attributed to influenza can be observed every year during the cold season, outside of any epidemic influence. It might be that he, considering the frequency of cases of la grippe, has taken some common catarrhal affection of the conjunctiva, for instance, as a manifestation of the epidemic disease; but there is no doubt that, with the exception of a few such cases, the disease imparts a special stamp to eye-affections which develop during the course of the disorder or after an apparent cure.

From the commencement of an attack of influenza there is observed a sharp conjunctivitis, whose origin may be traced to an intense catarrhal affection of the nasal mucous membrane. From this inflammation, or that of others of the membranes which line the neighboring sinuses, is derived an irritability of the trigeminus which may provoke pains that are referred to the back part of the orbit. Landolt has described a special form of conjunctivitis, affecting both the ocular and the palpebral mucous membrane, which he observed during convalescence from the disease. Here the superficial layers of the sclerotic participated in the inflammatory process, and assumed at times the aspect of a true episcleritis.

Mispelbaum[1] attributes certain neuralgias of the fifth pair occurring during the course of influenza to the state of irritation of those terminal branches that are situated in the sinuses in the vicinity of the nostrils. He affirms that the neuralgias of the supra-orbital and infra-orbital frequently fail to yield to electrical treatment.

During the epidemic of 1889–90, Berger observed a case of violent supra-orbital neuralgia of the right side. This was accompanied with photophobia, blepharospasm, and pericorneal injection, it being probably an affection of the frontal sinus. The conditions were relieved in a few

[1] Ueber Psychosen nach Influenza, Zeitschr. f. Psychiatrie, 1890, Fasc. 1.

days after a purulent flow from the right nostril had taken place. He considers those deep abscesses of the lids which Landolt and others have described as dependent on influenza to be due to affections of the frontal sinuses. Cases of supposed metastatic abscess of the orbit opening at the inner angle of the eye are considered by Berger to be produced by the continuation of the disease of the sinuses to the orbital tissues.[1] A case of Weichselbaum,[2] in which a young man was attacked with influenza and an abscess of the upper eyelid on the eighth day, is analogous to the above instance, and in fact is confirmatory; for, later, meningeal complications appearing, from which the patient died, the autopsy revealed that the sinuses were filled with pus.

According to Berger, these orbital abscesses may appear as late as several weeks after the termination of an attack of la grippe.

Referring to the epidemic of 1890, Gazis states that conjunctivitis, which was of rare occurrence in previous epidemics, was frequently observed in this, differing from the common catarrhal cases in the increased amount of secretion. The redness, however, of the palpebral and especially of the ocular mucous membrane was intense, and was accompanied with pain during the first days. It never lasted beyond from five to eight days, and he rarely observed the ecchymosis mentioned by other writers on the subject. The local treatment was the same as that indicated in catarrhal cases, besides the internal administration of quinine.

Galezowski recommends a careful examination, so as not to mistake it for the conjunctivitis which ushers in an iritis; for here the sharp pains present might make one suspect the presence of the latter if a careful examination of the iris did not show the easy contraction and dilatation of the pupil and the absence of posterior synechiæ. The invasion of the cornea in the course of influenza is fairly frequent, and in this connection Bergmeister considers an ulceration of a triangular or semilunar shape, accompanied with special zones of anæsthesia or blunted sensibility, to be pathognomonic. We have not, however, been able to confirm this in our own patients.

Hansen-Grut and Emmer have described a condition which they term *keratitis dendritica,* and which Eversbusch proposes to call *herpes febrilis corneæ.* In many cases this affection is accompanied with supra-orbital or ciliary neuralgia, and with herpetic vesicles on the eyelids.

Hirschberg mentions three cases of this form of dendritic keratitis occurring in influenza, appearing from three to five days after the commencement of the fever. There was no pain, only a moderate amount of irritation, and the ulcerations manifested themselves in the shape of ramifications. All the cases were cured at the end of a few weeks, leaving a slight cicatrix. The author attributes these ulcerations to herpes of the cornea.

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[1] Klin. Monatsblatt, 1891.

[2] Wiener Ges. d. Aerzte, January 30, 1890.

Keratitis punctata superficialis has likewise been observed, and, during convalescence from the disease, phlyctenular keratitis and episcleritis. The simple form of suppurative keratitis occurring in the course of influenza is found to be more obstinate. In a woman who every year was attacked with keratitis, which yielded easily to the usual treatment, we had occasion to witness the pains she experienced, the slow recovery, and, finally, the persistence of an albugo. In all her former attacks the transparency of the cornea had not been marred.

The muscle of accommodation frequently becomes affected. This is to be expected from an infectious disease which leaves the patients in such a debilitated state. With the recovery of the general strength the condition soon disappears.

Weeks, Éperon, Uhthoff, Badal, and others have confirmed this paralysis of accommodation. Sattler, however, believes it to be of rare occurrence, having seen but one instance among the many ocular manifestations that are due to the disease.

The external muscles are less frequently affected. There is no lack of facts, however, to demonstrate their having been attacked, for the above-named authorities report interesting cases of paralysis of the third and sixth cranial pairs. Sattler describes two cases of paralysis of the oculo-motor, of whose influenzal origin he was not, however, sure. Braunstein reports another. Arlt has observed a case of insufficiency of the internal rectus. Schirmer speaks of a case of total unilateral ophthalmoplegia. Greiff mentions paralysis of the right cervical sympathetic. Goldflam reports the following observations of nuclear paralysis consequent upon influenza. First, a physician sixty years of age was attacked, shortly after having suffered from la grippe, with ptosis of the left side and diplopia. Eight days later, drooping of the right upper lid, weakness of the legs, and convulsions "dans la station" appeared. There was a history of a chancre contracted twenty years before, and of epileptic attacks while pursuing his university studies. At the time of examination there were bilateral ptosis, immobility of eyeball, and contracted pupils. The irides reacted both to the stimulus of light and to the influence of accommodation. Vision for both near and far was normal. Rigidity of the fingers of the left hand, with temporary paralysis of the extensors of the arm, quickly followed by paralysis of the flexors of the right arm, gradually appeared. The triceps and the muscles of the lower part of the left side of the face lost their action. These symptoms revealed a lesion of the anterior bodies of the gray substance of the cervical cord and the nuclei of the floor of the fourth ventricle. Paralysis of the lips, difficulty of pronunciation, irregularity of the pulse, difficulty of deglutition, and death soon followed. Second, a woman thirty years old was attacked, without apparent cause, with pains in the nucha, ptosis, diplopia, paralysis of the upper and lower extremities, disturbances of speech and deglutition, dyspnœa, small and rapid pulse, chills, and, lastly, bulbar symptoms and salivation. The dyspnœa became

so pronounced that her life was despaired of. Nevertheless, it disappeared, deglutition became easier, and the patient got well.

The occurrence of papillitis in the course of influenza has been reported by various writers. Éperon refers to six cases of retro-bulbar optic neuritis, Bergmeister to two, and Hansen, Braunstein, and Weeks to one each. In Weeks's case a scotoma accompanied by various changes in the fundus of the eye was observed. No history of either tabes or syphilis could be elicited. There were no antecedents of alcohol or tobacco. Weeks insists that the scotoma is not identical with what is observed in cases of toxic amblyopia produced by alcohol and nicotine. The loss of sight came on suddenly after the attack of la grippe. Abstinence from alcohol and tobacco did not improve it. In a case of Landesberg's the affection of the optic nerve appeared fifteen days after the invasion of influenza.

As a rule, visual acuity and the field of vision diminish. These phenomena are accompanied with subjective sensations of light and violent headaches. At times the ophthalmoscope reveals a slight optic neuritis. No gross changes in the fundus of the eye, however, have been observed by Eversbusch, Uhthoff, Fuchs, and Bergmeister. In all, with the exception of the case mentioned by the last author, vision returned.

The amaurosis in such cases is undoubtedly produced by inflammation of the periphery of the optic nerve. This is proved by the progress of the disease, which commences by a narrowing of the peripheral visual field, and the sensation of pain that is produced by pressure of the globe backward.

A case of sudden amaurosis reported by Sedan differed from those produced by neuritis. Here the subject was seven years old. After three days of a bronchitis for which a sedative potion and one-quarter of a gramme of quinine per rectum had been prescribed, the patient was attacked with cephalalgia and delirium. On the following morning there was slight epistaxis, the temperature rising to 39° C. in the afternoon. At this hour the patient stated that he could not see. This statement was confirmed. On the following morning the fever moderated, but the child continued blind. He also experienced difficulty of hearing, this being due to the quinine. The drug was continued, and eucalyptus was ordered in addition. The child continued in the same condition for twenty-four hours, when after a long sleep he informed his mother that he could see, and asked for his toys. It was in this condition that Sedan saw him, and at first he was inclined to doubt the veracity of the accounts. The father,—a physician,—however, assured him that there was no doubt as to the child having been blind.

The writer has published an analogous case.[1] The wife of a physician was attacked by influenza. There was a temperature of $37\frac{1}{2}$° C., which increased until $40\frac{1}{4}$° C. was reached. On the following day subdelirium

[1] Crónica Médico-Quirúrgica de la Habana, 1892, p. 81.

appeared. Early on the following morning loss of sight ensued. At dawn the fever commenced to abate, and vision began to return until it reached its normal condition. No ophthalmoscopic examination was made, but it is not likely that there was any appreciable lesion in the fundus of the eye when vision was so rapidly recovered. Probably it was simply a passing spasm of the retinal vessels, similar to that seen in the cases communicated by Galezowski to the Biological Society of Paris.

There is no doubt of the optic neuritis of influenzal origin, which at times may be unilateral. The visual disturbance may be either permanent or temporary, and present all degrees of amaurosis. It appears from the third to the fourteenth day after the time of invasion, and is nearly always preceded by frontal and periorbital pains. The scotoma, which may be considered a toxic paralysis dependent upon influenza, is variable, and depends upon the part of the optic nerve affected. Landesberg has observed a case of central scotoma which gradually vanished, and Remak has seen one of the same kind for white and colors, which appeared four days after the commencement of the attack. Castens refers to a case of hyperæsthesia of the optic nerve which he attributed to la grippe. Berger, however, does not believe the condition to have been necessarily produced by an affection of the optic nerve, but looks upon it rather as a consequence of a concomitant affection of the nostrils and neighboring cavities that has been produced by irritation of the trigeminus.

The uveal tract is less frequently affected in la grippe. Among four cases of ocular manifestations due to la grippe, Braunstein reports one of serous iritis of the right eye of a fifty-three-year-old individual. It must be stated, however, that the patient had had a chancre twenty-three years previously. Adler and Fuchs each mention another case, both cases being accompanied with hypopyon, purulent hyalitis, and tenonitis. In both the pus perforated the anterior portion of the capsule of Tenon. Cultures made with this pus resulted in the cultivation of the pneumococcus of Fraenkel. Oeichelbaum Hosen relates the case of a child thirteen months old who, after an attack of iritis, had a purulent hyalitis which was due to cerebro-spinal meningitis.

Glaucoma has also been observed as a consequence of influenza. (Beaumont, Adler, Sattler.) The last author has witnessed the development of the condition during an attack of la grippe in persons of advanced age in whom the action of the heart was weak,—a fact that might well have been the direct cause of it.

Cases are mentioned of blepharitis, phlyctenular conjunctivitis, convergent strabismus, and blepharospasm. (Beaumont.) As Fage has conjectured in an extensive work, some are the direct consequence of the infection which brings about the febrile affection, and others are the result of the many modifications which it produces in the principal organs and apparatus of the general system.

No treatment has been established for these ocular manifestations dif-

ferent from that employed in analogous cases of different etiology. Whenever any special treatment has been proposed, it has failed to obtain preference.

Although the history of la grippe goes back to a very remote period, yet references to disturbances of the ocular apparatus have hardly been more than summarized. In 1833, in a memoir upon the disease, Pétrequin says, "I have studied in a special manner the pernicious influence of this epidemic on catarrhal affections of the mucous membranes, and particularly as regards ophthalmia. My purpose is to show the pathogenic relations existing between these various morbid states, and to make clear, through considerations of general and comparative pathology, the history of the catarrhal ophthalmia of this epoch." This reference of Pétrequin's is the most explicit of any made previous to the present time.

## OCULAR MANIFESTATIONS IN DYSENTERY.

The influence exerted by the diseased stomach and intestinal tract upon the ocular apparatus did not escape the observation of the ancients. In our days, by virtue of the physical means at our command for the exploration of the eye, the facts respecting these pathological relations have multiplied.

Investigation has demonstrated causes which are quite distinct from those that were previously believed in. Thus, in the case of an ophthalmia, for instance, it is no longer necessary to blame the stomach with the so-called "crudeza" (undigested food), nor the intestine with the "saburra" (foul intestinal residuum), worms, etc., as physicians in the days of Schmucker, Plenk, Richter, Scarpa, and Mackenzie were wont to do. Neither is it of any use to decry the value of counter-stimulants, among which tartar emetic occupied the first place. Reflex phenomena, however, which yield to opportune therapeutics directed against disorders of the digestive system are frequently observed in the apparatus of vision. Thus, Rampoldi has observed diminution of the power of accommodation during digestion. He believes that, as is the case in tobacco and alcohol amblyopia, the ocular phenomena are associated with, and are not secondary to, the pathological condition of the stomach and intestines. He, however, believes that the diseases of the latter organ produce less grave ocular manifestations. He supports this statement with the fact that tænia produces less reflex disturbance the further it is from the stomach. We must, however, remember that among the observations related by Rampoldi there is one of volvulus, in which, although it did not result fatally, the individual remained blind of one eye in consequence of atrophy of the head of the optic nerve. Here it is not stated whether the patient was examined before the intestinal disease was recognized. This is important, for the diagnosis of a monocular affection might well have been previously and accidentally determined, as so frequently happens. What Rampoldi has narrated, however, is

entirely in accord with what Lawson observed respecting a case of paresis of the muscle of accommodation appearing as a result of dysentery.

One can readily understand how easily the muscle of accommodation may be affected with paresis during the course, or as the consequence, of an infectious disease of the gravity of dysentery. For, not even considering the toxic effects of the infection, which are sufficient to weaken the whole organism, there remains the embarrassment to the functions of nutrition and the reparation of forces which must invariably result from the disorders of the digestive tract in general.

After the observations of Bouchard, says Grandclément, one can well explain the appearance of the visual disturbances of dyspeptic origin; we would add, also of those which originate in the intestine. In both they are simply the phenomena of auto-infection. The badly digested foods are converted in the alimentary tract into toxic alkaloids, which, when absorbed, carry their disturbing influences onward, and give origin to the thousand neuropathic forms of disease which alarm the patient so much, and of whose pathogeny the physician is unaware.

To recapitulate. Though the number of cases in relation to the ocular manifestations observed in the course of dysentery be limited, the facts above noted authorize us to divide the ocular symptoms into two groups,—reflex and those that are determined either by the special infection which produces dysentery or by such secondary infections as originate in them. In the first we may include paresis of accommodation, atrophy of the papilla, opacities of the vitreous, and false granulations. In the second may be placed conjunctivitis, keratitis, and choroiditis, produced by the passage of germs proceeding from the large intestine (at times suppurating or sphacelated) through the circulatory channels of the organism.

## OCULAR MANIFESTATIONS IN CHOLERA.

From the point of view of ocular complications, no distinction will be made between Asiatic cholera, cholera nostras, and cholera infantum. In the last two the causative agent is not always the same as in the first (the comma bacillus of Koch); but the analogy of the symptoms (digestive disturbance, algidity, etc.) in each indicates that the poison that is secreted by the different agents, such as the bacterium coli commune, altered meat, fermented milk, or certain medicines, as tartar emetic, etc., is probably identical in all.

In grave cases of cholera it is noticed from the first moment that the skin of the eyelids is bluish (cyanosis), and that upon account of difficulty in closing them they appear half open. If the patient is asked to close them, he can do it, but they soon open again. This is not only on account of weakness of the orbicularis muscle, but also because of contraction of the cellular tissue of the orbit, which causes the eye to retract, thus allowing the muscle to lose its point of support.

The lacrymal secretion is considerably diminished, so much so that, no matter how intense may be the pain experienced by the patient, he never weeps. The dropping of an irritating substance on the conjunctiva—laudanum, for instance—fails absolutely to provoke any hypersecretion of tears. (Graefe.)

During the asphyxic period of the disease the patient turns his eyes upward, so that either only a small portion of the cornea can be seen or else it is entirely covered by the upper eyelid. The bulbar conjunctiva, therefore, remains partly exposed, causing it to dry and become injected. Under these conditions Josreph has observed an ecchymosis which he believes is of fatal prognostic significance, for in twelve fatal cases the symptom was not slow in appearing.

Towards the end of the algid period a certain opacity which has not been covered by the upper eyelid has been observed situated in the superior portion of the cornea. If life is prolonged for one or two days longer, a superficial detachable eschar of a dark hue appears. The rest of the cornea, which up to this time has maintained its transparency, rapidly suppurates. Graefe admits that the keratomalacia observed in cholera is of a neuro-paralytic nature, but Berger believes that desiccation unquestionably plays an important *rôle* in its pathogeny. In grave cases of cholera the sensibility of the cornea is notably diminished. The existence of this condition has been confirmed by Campart and Saint-Martin.

The appearance of blackish spots in the sclerotic is a most unfavorable symptom. They are situated around the lower part of the border of the cornea. Their form is irregular, and they are disseminated; but at times they coalesce and constitute but one area. Boem and Graefe recognize in them the effect of desiccation of the sclera, which appears at a point that is situated opposite a similar area in the bulbar conjunctiva. Graefe has seen these spots make their appearance in the lower eyelids. Up to the present time their exact pathogeny is unknown, as no pathological examination of them has ever been made.

Enophthalmus, or retraction of the eyeball into the orbit, is also very pronounced in the algid period of cholera. This is due to a considerable diminution of liquid in the retro-bulbar tissues. It is not, however, in this disease only that the orbital cavity becomes empty in this manner, the phenomenon having been observed also in certain tumors. Delens mentions the case of an individual suffering from adenitis who had lymphatic tumors in the submaxillary region, the hard palate, and the pharynx, with axillary infarction. The orbits presented two symmetrical tumors of considerable size, which had grown in a period of from fifteen days to three weeks. Each tumor, which was indolent in nature, was of the size of a walnut, and elevated the upper eyelid. Vision was intact. Microscopical examination of the blood showed a considerable increase of the white corpuscles. The diagnosis of adenitis with lymphadenoma of the orbit and of the hard palate was made. Subsequently the patient was attacked

with cholera and recovered. Fifteen days later the tumors of the orbit and of the hard palate had disappeared. The submaxillary tumors remained, slightly infarcted. The axillary and inguinal swellings were reduced considerably in size.

During the algid period Graefe has found the pupils contracted. At the commencement of the disease they are dilated. Campart and Saint-Martin have frequently observed such dilatation. Graefe asserts, with sufficient reason, that the myosis seen during the algid period is produced by paralysis of the cervical sympathetic. The writer believes that a lesion of the sympathetic contributes to the retraction of the eyeball. Jacobson attributes the enophthalmus either to mechanical causes or to alteration in the blood or in the vessels. He does not, however, venture upon a thorough explanation of the question.

Bouchard thinks that the myosis is the effect of auto-infection which results from the accumulation of toxic products in the economy by reason of lesion of the kidneys, which prevents these organs from eliminating them. The result of his experiments, however, which consisted in injecting the urine from cholera patients beneath the skin of animals, refutes this theory; for, although he produced the symptoms of cholera in these animals, he did not obtain myosis.

During the epidemic of 1885 in Marseilles, Coste, while examining patients, observed that in the algid period the pupils were extremely variable in size. As these apparent pupillary conditions furnished him only contradictory indications of prognosis, he endeavored to find more certain types of pupillary reflex. From observations gathered from one hundred and twenty-seven patients each of whom he studied on an average for twenty-four hours, he arrived at the following conclusions. In the algid period of Asiatic cholera the state of the pupils, whether normal, contracted, or dilated, does not furnish any data towards forming a prognosis. The same rule, however, does not hold good regarding the pupillary reflex. When the reflex is present, the prognosis is favorable. When it is absent, the disease is fatal. In other words: 1. Whenever during the algid period of cholera, even though there be marked cyanosis, the normal or contracted pupils are susceptible of motion, one may form a favorable prognosis; or, what amounts to the same thing, if the pupils dilate on placing the eye in the darkness resulting from lowering or closing the eyelids, and return to their original diameter when exposed to the light, it may be said that the patient will escape this period, without being on this account protected from a relapse or from the complications of the period of reaction. 2. From the moment when the pupils are contracted and immovable, it may be asserted that the patient will succumb during the course of this period: this, he says, is true in spite of slight gravity in other symptoms, and even when the re-establishment of certain functions would seem to point to a happy termination. 3. If the dilated pupils are immovable (do not respond to the stimulus of light), the prog-

nosis is likewise fatal: here the case will terminate fatally in the course of this period. 4. When the pupils are sluggish, one may prognosticate a slow recovery of the patient, or perhaps that the duration of the recovery, independent of complications, will be proportionate to the pupillary activity of the algid period. 5. When the pupils are unsteady, it may be inferred, in spite of the slight gravity of other symptoms, that the pupils will finally contract and become immovable, and therefore that the patient will succumb during this period of the disease. The appearance of this pupillary sign, therefore, indicates, at least, paralysis of the reflex. In general, he says that if the pupillary reaction is weakened, a prolonged period of convalescence must be expected.

To Berger the opacities of the vitreous, and especially of the crystalline, prove that the uveal tract may be affected.

Ophthalmoscopic examination of the fundus of the eye during the algid period shows that there is a contraction of the retinal arteries, whose color appears of a dark red. In such cases slight pressure on the ocular bulb is sufficient either to provoke an arterial pulse or to exsanguinate the vessels of the fundus. These phenomena are considered to be the consequence of debility of the cardiac muscle and diminution of intra-ocular tension, this probably being dependent upon perturbation of the sympathetic. At the same time they are observed with the disappearance of the second sound of the heart and of the radial pulse. Contrary to what is observed in the arterial currents, the retinal veins retain their normal diameter and contain very dark blood. At times Graefe has observed the blood-currents to be interrupted in the veins of the retina. He has also seen the movement of small bloody cylinders in the retinal veins, as may be observed in some cases of embolism of the central artery of the retina.

Reference has already been made to the haziness of vision of which some patients complain at the beginning or during the course of the algid period of cholera. It is not known yet whether this ought to be attributed to weakness of the blood-currents or to ocular disturbances which have originated from the presence of microbes or their toxic products in the economy. Suppression of urine, in fact, is very frequent towards the end of the algid period of asphyxic cholera. This type of cholera predisposes to parenchymatous nephritis, which may bring about auto-infection by reason of the substances contained in the urine.

Frequently during the period of reaction of cholera a sharp conjunctival hyperæmia, which may degenerate into catarrhal conjunctivitis, has been observed.

The secondary complications, under the name of typho-choleraic, are lacking in any special influence in regard to the eyes, though Josreph maintains that, except in grave cases, where there is a state of mydriasis, the pupils are always contracted.

## OCULAR MANIFESTATIONS IN MALARIA.

Visual disturbances produced by malaria are mentioned in all works treating upon intermittent and pernicious fevers. The writer stated this in 1877 in his first writing on the ocular manifestations due to malaria. Poncet, one year later, likewise mentioned the fact in his interesting memoir on malarial choroido-retinitis.

Ozanam, Vaca, Berlinghieri, Morand in 1729, Arrachart, Pinel, Hildenbrand in 1821, Duboé in 1867, Testelin, Deval, Sloeber, and others, all point out the fact of disturbance of vision during the intermittent or pernicious paroxysm. They do not, however, go beyond this simple statement.

Later, ophthalmoscopic examinations made by Dutzmann in 1870 and by Königstein in 1875 gave only negative results. This examination of the fundus of the eye, as has always been the case, soon cleared up any doubts that might have been entertained by the first observers. Owing, however, to the circumstance that ocular disturbances of paludal origin can be observed only in certain regions, the number of cases collected up to within the last two decades is, proximately, very limited. When the writer published his first cases, in 1877, he was acquainted only with the observations of Gueneau de Mussey and Galezowski, which appeared in 1874. Poncet, in his memoir already referred to, observes that in such a complete bibliography as that of Nagel only a few documents relative to this study could be found. This was also spoken of in the Handbook of Saemisch and Graefe.

In more recent times, Kipp, Dehenné, Bard, Tangemann, Buren, Pieounow, Sulzer, and others have amply illustrated the subject of the ocular manifestations of malaria with clinical facts that confirm the symptomatology. Especially is this so in the description of its pathological anatomy which has been given by Poncet.

All modern authors agree that affections of the fundus of the eye—*i.e.*, neuritis, neuro-retinitis, and retinal hemorrhage—are the most frequent as well as the most important disturbances in the disease.

In situations where there is much malaria, such as Algeria, Poncet has found lesions that are appreciable to the ophthalmoscope in but ten per cent. of the cases that have been affected with malarial anæmia and cachexia. The microscope, however, nearly always exhibited alterations of choroido-retinal inflammation which were due to the paludal cachexia.

Lopez, of Havana, has found ocular manifestations of the fundus of the eye in four per cent. of cases affected with malaria.

Other authors have described manifestations of malaria in the conjunctiva, the cornea, and the iris. Tangemann reports three cases,—one of conjunctivitis with photophobia, another of iritis accompanied with photophobia and resistance of the iris to the action of mydriatics, and a third

of inflammation of the eyelids on the left side. All three yielded to quinine. The writer has reported a case of malarial conjunctivitis observed by Dr. Naranjo. Kipp, who divides the diseases of the eye that are due to malaria into two classes (one accompanying and the other following the paroxysm), places among the most frequent forms of the second class a superficial ulceration of the cornea. On the other hand, he has not found the form of serous iritis that has been mentioned by Picounow. Noyes speaks of the blunted sensibility of the cornea as a marked feature of malarial keratitis. He states that the membrane can be brushed with a feather without producing much disturbance. Van Millingen has described, as in influenza, a keratitis dendritica which appears during the attacks of intermittent fever. This variety of keratitis takes the form of serpiginous ulcerations with very fine prolongations. At first these appear as if raised from the surface, but finally they become ulcerated into a series of furrows. Its duration does not extend beyond three weeks. In grave cases it recurs with every new access of fever, which may be repeated during many months. The author apparently refers to the same lesion of the cornea as that observed by Kipp. Javal has met with the interstitial (parenchymatous) variety of corneal inflammation occasioned by malaria, and confirms the improvement that is said to be produced by quinine.

Sulzer has written an article, based upon the statistics found during the study of a large number of cases of acute and chronic malarial fever observed in the island of Java, in which he divides the ocular complications into two groups according as they accompany either the chronic or the acute form of the disease. In the first class, and in order of frequency, he places: 1, chronic optic neuritis with melanosis of the papilla: this is seen in the grave cases; 2, diffuse infiltration of the vitreous body; 3, multiple, punctiform, peripheral hemorrhages of the retina; and, 4, persistent amaurosis. In the second class he places: 1, periodical amaurosis and amblyopia without ocular lesion; 2, diffuse lesions of the fundus of the eye, predisposing to affections of the macula; and, 3, large peripapillary and macular hemorrhages in the retina.

Neuralgia of the trigeminus, especially of the supra-orbital branch, frequently takes the place of the paroxysms of an intermittent fever (Griesinger). The paludal origin of these neuralgias, however, has been very much doubted, and many cases have been diagnosed as malarial (outside of large malarial foci) which in reality were not of this type. Regarding this subject the writer has, with his colleague Dr. Madan, published a memoir with a view to demonstrate that we cannot specify as malarial a case of trigeminal neuralgia without the diagnosis having been preceded by a bacteriological examination of the blood and the discovery of the hæmatozoa described by Laveran as characteristic of paludism. These observations were based upon data that were collected during sixteen years in their clinic for diseases of the eye, and such cases as have been contributed by Dr. Coronado. In twenty-four thousand eye patients in the Havana

clinic, we have recorded two hundred and sixty-six cases of trigeminal neuralgia. Of these only thirteen were of malarial origin. For these reasons we have regarded as useless the administration of quinine for intermittent or periodical pains localized along the branches of the fifth pair. Periods of calm or of exacerbation of the pain at fixed hours are frequently observed in iritis of diverse etiology, as, for example, in the syphilitic or rheumatic varieties of the disorder. Especially is this the case towards the early morning, when cold aggravates rheumatic affections, or when the heat of the bed increases such pains as are of syphilitic origin. Moreover, it must be remembered that the affected eye is more or less fatigued or congested by the insomnia which accompanies nearly all diseases. The same thing happens in cyclitis following surgical operations, and in traumatisms, without any one being authorized to designate them as malarial so long as the examination of the blood does not justify such an assertion.

The relation of traumatism to primary disease, as studied by Verneuil, has also been described by Dehenné as regards the operation of cataract. The writer has published a case of intra-ocular hemorrhage in a woman who had suffered attacks of intermittent fever previous to the operative procedure. Delens has more recently strengthened this assumption with the authority of his observations.

Considering the manifestations of the fundus of the eye as those which are most frequently observed, the writer accepts, with Poncet, two forms of affections of the optic nerve that may be due to malaria: 1, an acute congestion, with slight elevation of the papilla, which is redder in tint than it is in its normal condition; 2, a chronic inflammation of the papilla, with venous congestion and a general dark appearance (melanosis). Around the papilla there is a gray œdematous haze or veil which obscures the arteries of the retina, whose diameter appears to be diminished. It is undoubtedly an intermediary stage between optic neuritis and neuro-retinitis. The field of vision remains normal, or, at most, a slight narrowing may be observed in the periphery.

The prognosis of paludal optic neuritis is generally favorable, for if one can combat favorably the malarial element with appropriate means, the acuity of vision is almost certain to be completely re-established. Cases are not wanting, however, in which atrophy of the papilla with complete blindness has supervened.

Sulzer has observed that in twenty per cent. of the cases acute malaria is accompanied with venous hyperæmia of the papilla. In addition there are itching in the eyes and a dazzling sensation, with photophobia. In these cases he has found hemorrhages in patches about the neighborhood of the papilla and the macula.

The intra-ocular hemorrhages due to malaria are at times numerous, as many practitioners have found; in fact, they are about as marked as cerebral hemorrhages due to the same cause are. The former appear disseminated about the periphery of the retina. In not a few instances either

embolism or cerebral hemorrhage is put down as the cause of loss of sight supervening suddenly as the effect of paludism. As regards the cases of sudden amblyopia and amaurosis without appreciable lesion of the fundus of the eye, Berger considers them to be due to a so-called toxic paresis or paralysis of the optic nerve. The hemianopsia and other accompanying cerebral symptoms are believed by him to be dependent upon embolus or cerebral hemorrhage.

In some grave cases of pernicious fever it has been noted that the patient has remained amaurotic for some days after the comatose state had passed off. The writer has observed some cases of amaurosis following paroxysms of comatose pernicious fever. Upon examining the fundus of the eye in these cases and finding there the characteristic signs of cinchonism, the amaurosis has been attributed to the effects of the drug rather than to paludism. The fact that these patients had taken enormous doses of quinine seemed to confirm the diagnosis. Before the ophthalmoscopic appearances of the amblyopia and amaurosis due to quinine had been as well described as they are to-day, the writer has seen these conditions mistaken for similar disturbances produced by malaria. This was due to the fact that, in countries where malaria prevails and counts so many victims, practitioners are afraid of too low a dosage of the drug against so formidable an enemy. Individual idiosyncrasy also counts for a great deal as regards tolerance to the remedy.

Instances of periodical amaurosis and amblyopia have been observed during the course of malarial affections. They may appear in paroxysms, and at times are associated with other cerebral symptoms, such as delirium and coma. In these cases, which are always grave, the amblyopia appears when the algid stage terminates. Again, as in other microbic diseases, it has been observed that the toxic paralysis manifests itself towards the end of the appearance of the general symptoms; moreover, according to various authorities, the duration of these attacks of periodical amaurosis is ordinarily from one-quarter hour to one hour. If they be frequently repeated, a permanent narrowing of the visual field may be observed, presenting in this a certain analogy with other forms of amaurosis (diabetic, uræmic) which terminate in partial atrophy of the optic nerve. Persistent disturbances of vision are observed only in grave cases of malaria, especially in the course of tertian. They are rarely seen in the quartan type of fevers. One observer reports periodical accesses of amaurosis without fever, which has been considered to be a symptom of a larval intermittent fever.

To recapitulate. Manifestations of paludism are rarely met with outside of large malarial foci. Optic neuritis, with its symptomatology, as described by Poncet, neuro-retinitis, and hemorrhages of the retina are the disturbances most frequently observed. Trigeminal neuralgia found in malarial foci, because of a certain and not well-defined periodicity, is frequently considered to be of paludal origin when in reality it is not. Conjunctivitis, keratitis, iritis, and other inflammatory disorders of the ocular

apparatus may be less frequently observed as due to malaria. Cases of amaurosis *sine materia* met with in the course of intermittent fevers, and in which neither the earlier nor the modern observers have been able to detect lesions, must be placed (Poncet) in the same group with malarial choroido-retinitis, in which there are emboli of melanotic leucocytes in the capillaries, which the microscope has been able to demonstrate.

## OCULAR MANIFESTATIONS IN DENGUE.

The careful study of dengue dates from the end of the last century. The principal foci have been two,—America and India. A great epidemic broke out in America in 1820 and lasted until 1828. It visited the greater part of the continent, and the Antilles. In spite of the small population of Havana at that time (fifty thousand souls), this place suffered severely from the epidemic. This is shown in documents to be found in the *Archivos de Sanidad*, which also indicate the hygienic measures that were adopted, besides giving a fairly good description of the disease.

The first great epidemic, which invaded the British Antilles in September, 1827, was known by the popular name of "dandy fever." Passing to the Spanish Antilles,—Puerto Rico and Cuba (March, 1828),—it received the appellation of "dengue," perhaps because of the stiff and measured gait the patients were obliged to assume during walking. Since then the expression "dengue" has predominated in successive epidemics, and has been made use of by the physicians of the French, English, and Spanish colonies, especially after having been accepted in 1869 for the nosological nomenclature that was formulated by a commission from the College of Physicians of London. In 1848 it reappeared in New Orleans, and later was observed in Havana. In 1860 it appeared in Martinique, and in 1864 showed itself in Cayenne. In 1856, in 1866, in 1876, and in 1880 it manifested itself in different parts of the United States and Mexico. The epidemic of 1848 invaded Brazil and Peru. In Asia, after the epidemic of 1789, it was observed in Hindostan in 1824, and again in 1826. In 1835 it visited Arabia and Lower Egypt, and many were the epidemics in those countries from that time until 1870. In 1871 it invaded a part of Zanzibar, extended to China, gained the Mediterranean, and reached Egypt. Still later, invasions have been successively noted in Ismailia (1877), in Egypt (1880), in Port Said (1883 and 1885), and in Cairo (1887). In 1888 it appeared in Syria and passed over to Asia Minor and European Turkey. Several epidemics have also been recorded in Senegambia.

Notwithstanding the great number of epidemics above mentioned, the writer has not heard of any special study of the ocular manifestations of the disease. They have, however, been observed, but, as in the case of influenza previous to recent epidemics, mere mention has been made of them. In fact, as a rule, they are so wholly analogous with those which are attributed to la grippe that an attempt has been made to establish the

identity of both of these infectious diseases by the similarity of the ocular symptoms. (Martiale.)

In dengue one of the most striking symptoms experienced from the commencement of the disease is pain. This is localized in the fingers, wrist, elbow, or knee, and at times in the nucha, in the lumbar portion of the spinal column, and, less frequently, in the epigastrium and gluteal region. Coincidently, or somewhat later, cephalalgia in the frontal region, accompanied with acute pain in the eyes and with photophobia, is complained of. (Otholendyl.) Krausenl has mentioned an instance of partial or total anæsthesia of the retina.

The face is red, and the eyes are brilliant and congested,—facts which might make one suspect yellow fever in a country where this disease is prevalent. Particularly is this so if the subject be a recent arrival from a cold climate. The doubt is, as a rule, soon dispelled by the absence of icterus of the conjunctiva or sclerotic. When both epidemic diseases, however, develop at the same time in the same region, as happened in New Orleans in 1848 and in Havana and Rio Janeiro in 1850, the presence of icterus in dengue has caused some practitioners to attribute the benignity of some epidemics of yellow fever to the fact that the disease was simply dengue.

In from twenty-four to thirty-six hours, and up to the fourth day, a bright red exanthem appears. This, which is similar to that of scarlet fever, has occasioned the name of "colorado," "fiebre roja" (red fever). The eruption is more pronounced on the face, nose, and skin of the eyelids, like that which is observed in erysipelas. The eyelids, as also the rest of the body, are affected with intense itching. At this time a second exanthem, which is somewhat different from the first, makes its appearance. (Hernandez Pojio, of Cadiz.)

At the time of the remission a profuse perspiration is apt to be observed. This seems to weaken the patient very much, leaving him in an alarming state of prostration. It rarely puts life in jeopardy, although some English physicians have mentioned cases of sudden death occurring during this period of the disease.

As this prostration disappears, as in la grippe, the patients notice diminution of sight. This is due to paresis of the muscle of accommodation, which is so frequently noticed in all phases of general adynamia.

Among the varied ocular nervous phenomena occurring in dengue, hyperæsthesia of the retina has been observed. Vernani has described it as a special alteration of vision in which all objects are characterized as being of a yellow or a red color. The six patients whom this author saw presented this symptom,—saw all objects as though they were illuminated by the glare of a great conflagration. The ophthalmoscopic examination did not reveal any lesion of the fundus of the eye.

The articular and the muscular pains are constant, and to the latter type must undoubtedly be referred those which are experienced by the eyes during the slightest movement of the eyeball.

This ocular pain, and also that of the articulations and muscles of the body, have some similarity to the pain of acute rheumatism. They cannot, however, be regarded as of the same origin with that which is observed in that diathesis. Rochard calls the pains which proceed from the articulations, arthralgias. He finds that they have great resemblance, without bearing any relation, to the rheumatoid pains of syphilis.

Conjunctivitis has been observed during the course of the disease. As in influenza, suppurative keratitis has either appeared as an extension of the conjunctival inflammation or been recognized from the beginning.

Iritis has been noted by several authors. No details, however, which might justify a diagnosis of true inflammation, instead of a simple congestion of the iris which is so frequent in cases of conjunctivitis and keratitis, have been given.

The irido-choroiditis of which treatises speak might inspire the same doubt, although in infectious diseases, such as dengue and influenza, there is no reason why infection or its effects should not be limited to certain regions.

According to Mahé, cephalalgia accompanied with periorbital oppression was the predominant symptom of the Cadiz epidemic. In the epidemic of India in 1872, congestion of the bulbar and palpebral conjunctiva, particularly marked on the lower eyelid, together with a sharp cephalalgia, ordinarily supra-orbital, was noted by Wise. Nearly always the eyes are watery and injected and of a bright scarlet color. Their painful condition is increased by simple pressure over the eyelids. To the patient they feel as if they were enlarged and as if they would jump out of their orbits. These symptoms occasion extreme anxiety.

Mahé adds that among the complications and exceptional signs, amaurosis, partial paralysis, and a kind of mental weakness, which is quite marked, have been noted in previous epidemics.

De Brun, in his description of the epidemic observed in Beyroot, speaks of the frontal cephalalgia as the principal symptom. He says that the pain is frequently localized in the lower part of the forehead, on a level with the superciliary arch, where it remains fixed and immovable for two or three days, becoming intolerable when the patient moves about: other patients complain of an extremely painful sensation in the back part of the eyeball or of the orbit, which is similar to that which might be produced if the eyeball, having become very voluminous, were to be strongly compressed by its containing walls. The patients avoid the light, which seems to increase their annoyance.

As dengue is regarded by many authorities, especially Klassen, as a special eruptive fever, it is evident that the ocular apparatus cannot be exempt from the complications which accompany the majority of eruptive fevers; that is, they are the result of secondary infection or of such as originates in the special germ which produces the principal disease. This idea has gained positive value since Panas, upon enucleating the eye of

an individual who had suffered a short time previously from typhoid fever, found a focus of suppuration at the apex of the orbit, in which the bacteriological examination demonstrated the presence of the Eberth-Gaffky microbe.

## OCULAR MANIFESTATIONS IN YELLOW FEVER.

These have hardly been mentioned in any monograph that has been written upon this subject. After describing the commencement of the fever, and the general reaction or "coup de barre" (lumbar pain), characterized by agitation in the epigastrium and tumultuous beating in the cœliac region, passing note is made of the dilatation of the pupil, which, however, is so marked that it gives a vague and undefined expression to the physiognomy very similar to that seen in the face of an inebriated person.

At the end of the first day or later (Bérenger-Féraud says on the second day) attention is called to the state of hyperæmia of the face and eyes. The eyes, which appear red, lacrymosed, and brilliant, acquire considerable importance in the symptomatology of the disease. Bérenger-Féraud has found that during the second period of the affection, which is known as the period of localization, and which commences with a remission, the coloring of the eyes diminishes simultaneously with that of the skin. He has also noticed that the manifestations of icterus make their appearance, these commencing at the sclerotic.

Speaking of the hemorrhages in yellow fever, Naegeli says, "The ocular and auricular hemorrhages are extremely rare, and the loss of blood is insignificant." Bérenger-Féraud states that "the brilliant eyes, the injected conjunctiva, which varies from pale to bright rose, do not present that hyperæmic aspect which is so marked during the last stages of the affection." In this disease, the latter author observes, there exist certain anomalies which must warn us against forming too absolute conclusions. The original congestion may become inflammatory, in which case the coloring ceases to have any prognostic importance. More recently the same author, in a work upon "Yellow Fever in Martinique," says that the second period communicates a greater intensity to all the ocular symptoms. He adds that this intensity diminishes with the remission of the fever, but notes that if the conjunctiva becomes obscured and takes the icteric hue the case presents greater gravity. In some grave cases he has observed ocular hemorrhages which have produced subconjunctival effusion or flowing of the blood into the neighborhood of the commissures. Griesinger is the only author who mentions the diminution or loss of sight, when he says, "Amblyopia and amaurosis are of rare occurrence in yellow fever."

It will thus be seen that these authorities have mentioned only external manifestations, which have been confined generally to the cornea and the conjunctiva.

In 1881 the writer published the following cases of partial or total loss of sight:

CASE I.—A cadet was admitted to the "Quinta de Garcini" on August 24, 1879, in the second day of yellow fever. This young man, an Andalusian, arrived from Europe in perfect health. The march of the disease was insidious; the temperature was 39° C., the pulse averaged 100, and there was a small amount of albumen in his urine. He was given an emetic of ipecacuanha. On the following day the symptoms continued without change. Forty grammes of sodium sulphate were then administered. The third day, the stupor, albuminuria, and very high temperature continuing, an enema of castor oil was given, and he was ordered to take tartaric acid lemonade *ad libitum.* On the fifth day signs of undoubted gravity—anuria, hiccough, delirium, and loss of sight—appeared. Upon consultation, revulsives, tonics, and diuretics were prescribed. Twenty-four hours later the urinary secretion was re-established and sight returned. The patient recovered without complications.

CASE II.—A Galician sailor, aged thirty five, contracted the disease on board ship. He was admitted to the Hospital de Gibara. On the following day the presence of a large amount of albumen in the urine and high temperature caused an unfavorable prognosis. Upon the next day the gravity of the case increased. There were hæmatemesis, anuria, and amaurosis. The patient died during the night. An ophthalmoscopic examination was not made.

CASE III.—A priest, recently from Gerona, remained several hours at the grave of a fellow-priest who had just died of the disease. On the following day the patient took to bed. The pupils were dilated, the face was injected, and the tongue was elongated. Pulse averaged 108, and temperature 37.8° C. There was but little urine. He was given a cathartic of castor oil. In the afternoon he had black vomit. Cold compresses were then applied to the forehead. The following day the cathartic was again administered. Upon the second day the eyes were freshly injected and lacrymosed. Delirium and restlessness now set in. There was but little urine. The pulse averaged 110; the temperature was 39.9° C. Anuria and loss of sight came on. The conjunctiva was injected and jaundiced. The pupils were not contracted, though an ophthalmoscopic examination was impossible without the use of atropine. Twenty minutes after the employment of the drug the fundus of the eye was with great difficulty examined and found to be normal. The following day there was a remission of the symptoms, but the amaurosis persisted. Upon the morning of the fourth day melanic vomiting appeared, soon followed by fetid stools, syncope, and death.

The aspect of the eyes is very characteristic: it constitutes the most apparent part of what has been termed by Roux, Bérenger-Féraud, and Jaccoud the mask of the disease. From the moment of invasion of the disease the eyes are brilliant. At times they are tearful, and nearly always they are sensitive to light.

The ocular conjunctiva appears finely injected. This is doubtless

caused by paralysis of the vaso-motor nerves, which, according to Bemiss, produces the bright coloring of the face as one of the first effects of the yellow fever poison. As the remission approaches, the conjunctival hue changes, so that if the attack is light the color fades without becoming yellow. At other times, however, it assumes a deep yellow tint from the time of invasion. If the case is a grave one, the conjunctiva remains a long time red or dark red. When this fades, it soon takes the icteric tint, which as the disease progresses becomes increasingly accentuated. At times the conjunctival injection is so pronounced from the first stage of the fever that during the remission, or what is known by Roux as the fourth period, it presents the aspect of a true inflammatory hyperæmia, which Dutroulau compares to the condition ordinarily seen in the eyes of the rabbit. In some grave cases, when the disease has advanced, subconjunctival effusions of blood may be observed. True ecchymoses, and even hemorrhages which produce a gradual flow of blood at the commissure of the eyelids, may appear.

During the terminal period, or what Roux terms the fifth period of the disease, which generally lasts about twenty-four to thirty hours, and corresponds to the fourth to tenth day of the disease, the most marked symptom is jaundice. This, which first appears in the sclerotic and is accompanied with lacrymation, soon afterwards shows itself in the skin. Sometimes the icterus of the sclera and of the skin may be absent during life, especially that of the latter. It is sure, however, in such cases to appear a few moments after death. Dutroulau states that he would not assert that a patient had died of yellow fever if at the autopsy he did not find at least the icterus of the cellular tissue.[1]

From the period during life in which the icterus is observed, Jaccoud infers that the organic changes which produce it appear prematurely, these manifesting themselves earliest in the hepatic apparatus. The icteric tint observed in the fifth period of the disease is more variable, and is not always as well marked as might be supposed from its designation. The characteristic hue may be light yellow or olive green. In the majority of cases it approaches more nearly to the first hue. Independently of this jaundice, another may appear. This has been called false jaundice, and authorities are not in accord regarding its origin and prognostic importance.

Although the duration of yellow fever cannot be fixed in any precise manner, most authors agree that five or six days constitute its average length. Completely cured patients may, however, for a long time present the icteric hue of the sclera and of the skin.

In some grave cases not only may the conjunctiva be attacked by true inflammation, but this may extend to the cornea, which may become ulcerated. In the author's clinic for diseases of the eye, in Havana, there have been persons who were affected with ulcerative keratitis that super-

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[1] Pathologie Intertropicale, p. 197.

vened in the course of yellow fever. In one of the hospitals of the same city he has seen a case of panophthalmitis appear during convalescence from the general disease. Whether this inflammation of the eye was an extension of one that was primarily seated in the conjunctiva or cornea, or whether it might at the present day be explained by ocular infection, in the same manner as has been observed in typhoid fever, tuberculosis, and gonorrhœal rheumatism, are interesting conjectures.

The pains experienced in yellow fever are characteristic. The cephalalgia is nearly always limited to the fronto-orbicular region, and is felt in both eyeballs. It is fixed, intense, and persistent, even to the point of causing the patient to cry out. This and the lumbar pain ("coup de barre") bring about the accompanying restlessness, which is relieved only by frequent changes in position.

The patients frequently attribute the sensation of glare in the eyes to the cephalalgia, but this may be produced by the conjunctival hyperæmia, which is in itself sufficient to give rise to photophobia. At the beginning of the disease the supra-orbital headache reaches its maximum, diminishing with the increase of the fever. It does not possess much prognostic significance, since Roux, out of twenty-four cases which resulted fatally, found it to be slight in nine, intense in twelve, and absent in three instances.

From what has been said, it may be inferred that in yellow fever there are ocular manifestations which depend directly upon the action of the specific poison. Among these may be classed the icterus of the conjunctiva and the neuralgia of the ophthalmic branch. On the other hand, the conjunctivitis, keratitis, panophthalmitis, and the hemorrhages of the ocular bulb respond to the disturbance which the source of infection produces in the economy, thus rendering it more apt to receive other infections. The amblyopia and amaurosis without any lesion that is appreciable to the ophthalmoscope are due, so far as the writer has been able to ascertain, to toxæmias that originate from disturbances of important functions, such as that of the kidney. As a rule, these ocular symptoms are associated with suppression of urine, and are accompanied with violent delirium, as a natural consequence of the concomitant uræmia.

During convalescence, secondary infections of diverse origin may be observed. These can be recognized as superficial by keratitis, or else as deep by panophthalmitis.

To recapitulate. The ocular manifestations of yellow fever are of two kinds, and correspond to different periods of the disease. The first are of a congestive nature, and are localized in the conjunctiva, particularly during the first period, and, when intense, pass on to the second period with the characteristics of hemorrhagic icterus. The second is characterized by loss of sight, which is more or less rapid in its appearance. It is accompanied by anuria, and may be considered, by reason of the absence of intra-ocular lesions and their easy re-establishment, to be a characteristic sign of uræmia.

# THE OCULAR MANIFESTATIONS OF HYSTERIA.

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No one now ignores the important position (chiefly won by the labors of Charcot and his pupils) occupied of late years by hysteria in the pathology of the nervous system. We no longer regard this affection as strictly confined to the female sex, since, according to the statistics of Pitres, P. Marie, and Souques, its graver forms are seen quite as frequently in men as in women. Moreover, although it usually singles out adolescents between ten and twenty, it may affect persons of all ages. In its etiology hereditary nervous and moral influences play the chief part, but its determining causes are manifold, including, for example, traumatism and toxic agents. We no longer look upon hysteria as a disease distinguished chiefly by simulation and moral perversion. From this disease without apparent lesions,—a psychic disease, if you will,—even though there can be discovered no anatomical changes in the nervous elements or in the structure of the apparatus affected by it, there may result paralyses and contractures quite as lasting and, on account of the disabilities which they produce, quite as serious as the paralyses and contractures that accompany lesions which are both material and destructive.

Hysteria assumes various aspects, from the moral perversion that transforms the sufferer into a kind of unconscious automaton, apparently calling for the aid of the alienist, to that of a local deformity often considered and treated as a surgical disease. The longer we study them the more fully do we recognize the fact that, if hysterical subjects are not the simulators we believed them to be, hysteria itself can so well imitate a number of organic diseases as to deceive the clearest heads. This fact is at once the explanation and the extenuation of errors over which we have finally triumphed.

In the midst of this symptomatology, so varied, so changeable, and so deceptive, the ocular manifestations are singularly constant and character-

istic. Indeed, these ocular signs form an element in diagnosis whose importance was often pointed out by Charcot. I should not have been criticised by the great master, whom science has just lost, for asserting that during his studies of hysteria—especially of those forms seen in men and in traumatic cases—he found the eye-symptoms a never-failing guide in unmasking the neurosis and establishing its identity, even in the presence of the most conflicting testimony.

The ocular manifestations of hysteria should not only interest the ophthalmologist and the physician, but should merit the attention of the physiologist in the highest degree. They shed a new light upon the function of the visual apparatus in situations where our present theories give us little assistance. Charcot often insisted upon the necessity for investigating hysteria from a physiological and not from an anatomical stand-point. A local hysterical affection may imply the intervention of anatomical centres, both multiple and distinct. In a general way we can say that hysterical symptoms do not indicate the lesion of a muscle, of a nerve, or even of a nervous centre, but point to the disease of a function.

On the other hand, Pierre Janet, in certain remarkable studies of the subject, has given prominence to another fact, that hysteria is especially characterized by disaggregation of the personality, meaning by this term the disassociation not only of those functions that distinguish the personality, but also of those elementary faculties that make up each function.

It follows from this dual characteristic that when hysterical affections are carefully studied they present physiological phases so delicate and so varied that the most profound student cannot conceive of anything with which to compare them. When the prejudice shown towards everything that pertains to the study of hysteria disappears, when we no longer refuse to avail ourselves of this inexhaustible storehouse of facts, the physiology of the brain and of the senses will have entered upon an era of rapid progress.

Hysterical affections are of two kinds. Some are permanent, having a duration more or less long,—sometimes as long as life itself; other manifestations, such as convulsive attacks and hypnotism, are more transitory. The permanent affections of vision, *the ocular stigmata of hysteria,* constitute a symptomatology which is absolutely distinct and unvarying. If, however, we confound with these clinical signs, properly so called, those which pertain to suggestion and hypnotism, we at once alter their true meaning and diminish their semeiological value. In this study the writer will consider only the permanent affections of sight,—the ocular stigmata of hysteria. He will add a few words regarding those symptoms that accompany or follow attacks of the disease, but will eliminate everything that relates to hypnotism and suggestion. Not that he denies the utility or interest of these phenomena, but they belong to a class by themselves, and require for their study a mind especially fitted for that kind of experimentation. Within a territory purely clinical they may serve as control experiments to determine whether, for example, such or such a symptom is or is not

hysterical in character. According to Möbius and Babinsky, it is characteristic of hysterical affections that they can be reproduced by suggestion. A patient was lately presented to the Medical Society of the Paris Hospitals who had a divergent strabismus which was regarded as of hysterical origin. Babinsky denied that it was hysteria, on the ground that, while it is possible by suggestion to produce a convergent strabismus, it is impossible thus to produce a divergent one. The autopsy, made shortly afterwards, showed that he was right. The strabismus was the result of a nuclear lesion and a neuritis of the third nerve.

The ocular disorders of hysteria are of two kinds,—first, those that affect the apparatus of special sense, and, second, those that concern the ocular muscles. In spite of the close relationship existing between these two groups of phenomena, their study should be pursued in two distinct chapters. The first of these will be entitled *hysterical amblyopia;* the second, *affections of the motor apparatus of the eyes.*

Variations in the general sensibility, of much less importance, do not require special notice, and will be considered in connection with hysterical amblyopia or asthenopia.

### HYSTERICAL AMBLYOPIA.

Cases of hysterical amblyopia were recognized at a very early date by Charles Lepois (1618) and Carré de Montgeron (1745). The first important work on the subject was that published by Hocken,[1] who thought that the amaurosis was always produced by a contracture of the orbicularis,—an opinion combated by Landouzy.[2] Briquet[3] made a more complete study of hystero-visual affections.

The amblyopia described by von Graefe[4] under the name of "True Anæsthesia of the Retina," and by Schweigger[5] under that of "Amblyopia of the Visual Field," is nothing else than hysterical amblyopia. Von Graefe insists upon the concentric limitation of the visual field. He believes that the visual defect is the result of an interruption of conduction between the bacillary layer and the nerve-fibres,—in other words, that it is a peripheral affection. Although this interpretation is false, the term "anæsthesia of the retina" hardly merits the criticisms to which it has been subjected. Doubtless the disease is of central origin, but as long as neurologists refer to hysterical "anæsthesia of the skin," hysterical "anæsthesia of the pharynx," etc., we are justified in speaking of "anæsthesia of the retina." This designation is in all cases to be preferred to that of "hyperæsthesia of the retina," proposed by Steffan.[6]

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[1] Schmidt's Jahrbücher, 1844.
[2] Traité complet de l'hystérie, 1846.
[3] Traité de l'hystérie, 1859.
[4] Klinische Monatsblätter für Augenheilkunde, 1865.
[5] Handbuch der Augenheilkunde, 1871.
[6] De l'anesthésie de la rétine Bericht der ophthalmol. Gesellschaft, Heidelberg, 1873.

German authorities who, following von Graefe, refer to anæsthesia of the retina do not sufficiently insist upon its connection with hysteria. Förster[1] remarks that this neurosis plays an insignificant *rôle* in the production of retinal anæsthesia. The hysterical character of the visual defect is, however, clearly demonstrated by Charcot[2] and by Leber,[3] who place the seat of the disease, not in the retina, but in the brain. The dyschromatopsia imperfectly recognized by Briquet has been clearly and fully described by Galezowski[4] and Landolt.[5]

Among more recent works may be mentioned those of Horstmann,[6] of Wilbrand,[7] and of Frankl-Hochwart and Topolanski.[8] In France have been published the thesis of my pupil Hittier,[9] a very complete study by Gilles de la Tourette[10] in his treatise on hysteria, and the thesis of Pansier.[11]

Disorders of vision in hysterical subjects present practically the same well defined characteristics that the writer described[12] in 1886. The old name, anæsthesia of the retina, has been retained because the writer was not then certain that this form of anæsthesia is peculiar to hysteria. As a matter of fact, it was found associated with certain organic lesions, with traumatisms, and with poisoning. It was also seen in children as a transitory manifestation connected with marked hysteria. Now we know that it may be associated with organic lesions without being identified with them, —that is, may accompany such physical influences as injuries without losing its separate hysterical character; we are now aware that, apart from its more conspicuous signs (*la grande hystérie*), there are many local symptoms, more or less transitory, of which children are frequently the subjects.

It is becoming more and more certain that an amblyopia characterized not only by contraction of the visual field, but by other symptoms about to be detailed, is peculiar to hysteria. The name hysterical amblyopia is thus preferable to any other.

The essential sign of this affection is *concentric contraction of the field of vision*, to which may be added, in certain cases, a particular form of *dyschromatopsia*. One peculiarity of this anæsthesia of the retina is that it is accompanied by spasm of the accommodation, which, differing from a true

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1 Graefe und Saemisch's Handbuch der gesammten Augenheilkunde, vii. S. 145.

2 Leçons sur les maladies du système nerveux, 1868.

3 Amblyopia hysterica, Archiv für Ophthalmologie, 1869

4 Les altérations du nerf optique dans les maladies cérébrales, 1866.

5 De l'amblyopie hystérique, Archives de physiologie normale et pathologique, 1875.

6 Ueber Anesthesia Retinæ Deutsche medicinische Wochenschrift, xx., 1885.

7 Ueber neurotische Asthenopie und die sogennante Anesthesia Retinæ, Archiv für Augenheilkunde, xii., 1893.

8 Zur Kenntniss der Augensymptome der Neurosen, Beiträge zur Augenheilkunde, xi., 1893.

9 De l'amblyopie liée à l'hémianesthésie et spécialement de l'amblyopie hystérique, 1886.

10 Traité clinique et thérapeutique de l'hystérie, 1891, pp. 321–432.

11 Manifestations oculaires de l'hystérie, Thèse de la Faculté de Montpellier, 1892.

12 Anesthésie de la rétine, Annales d'oculistique, Mai–Juin, 1886.

amblyopia properly so called, forms an integral part of the hysterical affection,—that is to say, the accommodative failure plays a certain part, not only in the visual affection, but also in the muscular defect. It is not a mere coincidence, but constitutes one of the most distinctive traits of hysterical amblyopia. From this contraction of the amplitude of accommodation result two other symptoms,—*monocular polyopia*, which the writer described [1] in 1878, and *micro-megalopsia*. These two symptoms are readily demonstrated, and in doubtful cases permit us to recognize at once the nature of the defective vision.

(*a*) *Contraction of the Visual Field.*—By contraction of the field of vision we usually mean a contraction determined by a white object on a black background. It refers, consequently, to an insensibility on the part of a portion of the field to white light, and the affection is not (as some authors, especially Steffan, assert) characterized by a hyperæsthesia of the retina. While it is true that the amblyopia is habitually accompanied by photophobia, and that a strong light sometimes exaggerates the contraction, yet it is not the less certain that the distinguishing mark of this affection resides in a diminution or in the complete abolition of the faculty of receiving luminous impressions, as may be proved by a quantitative exploration with the photoptometer.

This insensibility to white light argues ordinarily an insensibility to other light rays, and, as a matter of fact, contraction of the field for white is usually accompanied by a similar contraction for colors. This rule is not without exception, and we find that one of the most curious manifestations of hysterical amblyopia lies in an occasional reversal of this rule. Sometimes the field for white is much more restricted than that for certain colors.

The contraction of the visual field is concentric,—that is to say, the insensibility develops from the circumference to the centre in such a way that a graphic drawing of the contraction would be represented by an almost circular line. The writer says almost circular, for one must not take in its literal sense the term *concentric* limitation. The contraction is quite noticeable on the temporal side, where, normally, the field is largest, but it is often most restricted in the nasal or superior portion. With these slight variations, we may regard the concentric limitation of the visual field as one of the most important characteristics of hysterical amblyopia. In this connection the writer desires to speak of certain charts which, under the pretence of precision, entirely misrepresent the facts of the case. The contraction of the field of vision shows in many instances a certain inconstancy which goes under the name of "oscillating visual field." It may diminish in size during an examination under the influence of efforts at fixation or through fatigue. Thus, if one methodically measures several meridians, necessitating an examination more or less prolonged, it may

[1] La polyopie dans l'hystérie, Annales d'oculistique, Mai–Juin, 1878.

easily happen that at the end of the time so occupied the chart will present all sorts of irregularities, according as these particular meridians have been the first examined or the last. Finally, Förster[1] has shown that if the fixation-point be moved from the centre to the periphery the field in each of the meridians will be larger than when the object is moved from the periphery to the centre.

The extent of the contraction varies greatly. On the temporal side the reduction in size sometimes amounts to only ten or fifteen degrees; and since the normal field in certain subjects is only seventy-five or eighty degrees, it is difficult to say from this symptom alone whether there is amblyopia or not, especially if contraction exist in both eyes. It is then that the concomitant disturbance of accommodation will dispel doubt.

Peripheral vision is sometimes entirely abolished, perception being limited to central vision and extending only eight or ten degrees from the fixation-point. This condition may persist for years without modification. When central vision is itself affected, amaurosis becomes complete, and the insensibility is sometimes as profound as in the most advanced degenerations of the optic nerve: no light, however powerful, conveys any luminous impression.

Among the causes which may modify the extent of the contraction should be first noted convulsive attacks, which exaggerate the amblyopia to the point of producing a temporary amaurosis. We find, then, in general, the visual field more contracted if the patient has had convulsive seizures prior to examination.

I have observed that in certain subjects we may modify the extent of the visual field by acting upon accommodation with spherical glasses. In some patients, in whom vision was markedly reduced not only by the disorder of accommodation but also by anæsthesia of the retina, I have seen the visual acuity return to such a degree that it was impossible to explain the amelioration by the laws of refraction alone. Unquestionably we can modify peripheral and central sensibility in hysterical amblyopia by means of the accommodation. It is true that we sometimes obtain the same result by the application of neutral glasses to the eyes. It would not be necessary to believe this to be due to suggestion,—which may nevertheless exist in some instances,—or to some mysterious action of the glasses. We must take into consideration the instinctive tendency to relax or contract the accommodation when glasses are placed before the eyes. I believe, with Förster, that the extent of the visual field in retinal anæsthesia is modified by the action of atropine.

The converse is equally true. Excitation of the retina by light increases contracture of accommodation, especially when there is intense photophobia. We may, perhaps, explain the paradoxical fact that the visual field is some-

[1] Mesure du champ visuel dans l'anesthésie de la rétine, Bericht der ophthalmologischen Gesellschaft, Heidelberg, 1871.

times more extended with a reduced light than with an intense light by remembering that retinal sensitiveness is sometimes associated with spasm of accommodation. Smoked or blue glasses also increase the extent of the visual field, as has been for a long time noted.[1]

Morawesik and Freund have pointed out a curious peculiarity,—that cutaneous excitations modify the extent of the visual field. The writer has been able to show that puncturing the skin, on no matter what part of the body, increases the extent of the visual field until in certain cases it renders it normal. Excitation of one half of the body may modify the visual field of both eyes. When the amblyopia is unilateral, cutaneous excitation produces sometimes a transfer, the narrowing of the field disappearing in the affected eye and developing in the healthy eye. Cutaneous excitation is without effect in the presence of cutaneous anæsthesia.

In a recent report Le Dantec[2] stated that cutaneous excitation in hysterical patients produces subjective sensations of color. In one patient, the eyes being previously covered, pinching the skin on the border of an anæsthetic area gave a sensation of green; excitation by heat caused a sensation of red.

(*b*) *Dyschromatopsia.*—The narrowing of the visual field, which explains the insensibility to white light, constitutes the fundamental fact of hysterical amblyopia. The disorder of vision as regards colors is a superadded fact which is not constant.

Like insensibility to white, insensibility to colors is first manifested at the periphery of the visual field by the narrowing of the color-field. Again, it is often difficult to diagnose dyschromatopsia by the narrowing of the field for colors alone, by reason of individual differences which are found in the normal state; because of those which depend on the light and the objects employed; and, finally, upon account of the narrowing due to the insensitiveness to white light.

When the field for white is reduced to ten or fifteen degrees, the color-field may be contracted in the same proportion without any dyschroma-

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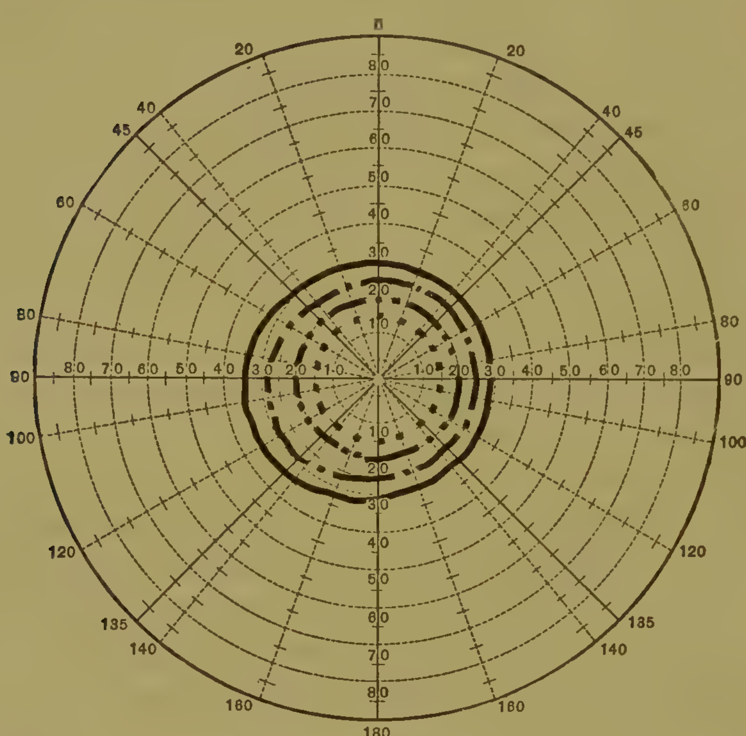
[1] I have recently observed a remarkable fact which demonstrates, on the one hand, the influence of light upon the development of spasm of accommodation, and, on the other, confirms the reports that connect the contracture with narrowing of the visual field. A boy, ten years of age, whom I am attending for slight accommodative spasm and a slight narrowing of the visual field, exhibited so little asthenopia as not to necessitate the interruption of his studies. He passes the greater part of the day out of doors, exposed to the sunlight. His sight is becoming rapidly affected. He asserts that intense light becomes painful; also that it causes vertigo and nausea. He is better as soon as he is brought into a dark place. He cannot go out after four or five o'clock in the evening. He was brought to me on August 21, and I found a double spasmodic myopia of eight diopters, the eye being slightly hypermetropic, with a visual field that was contracted to from five to six degrees. I prescribed atropine for eight days, and ordered him to wear tinted glasses. He was again seen on October 9. The spasmodic myopia and the excessive contraction of the field had entirely disappeared on the left side. The field is limited to about forty degrees in the right eye, and there is some spasm of accommodation.

[2] Société de biologie, Paris, July 8, 1893.

topsia. The circles that limit the different color-fields are close to one another, but preserve the same relative position that they do under normal conditions. (See Fig. 1.) It was in accordance with these facts that Landolt figured the charts which he publishes as characteristic of hysterical amblyopia, and which, on the contrary, are simply those cases in which there is no dyschromatopsia.

The patient may even cease to recognize colors, owing to the increase of the amblyopia, in the same way, for example, that a person may acquire cataract without having his color-sense especially affected. The loss of

FIG. 1.




Narrowing of the visual fields for white and colors. The circles have the same arrangement as in the normal condition.

White ———— Red ——▪▪—— Blue ——▪—— Green . . . . . .

color-vision occurs in the same order as in dyschromatopsia, properly so called. The perception of dull colors weakens first.

That we may be able to affirm the presence of dyschromatopsia from the contraction of the color-field alone, it is necessary that the latter should be well marked relative to the contracted field for white, and this, in practice, is a very difficult matter. There is another means, however, by which we can early and easily demonstrate the presence of a dyschromatopsia,—by examining the field of vision. This test depends upon a variation in the size of the field for white relative to the color-fields, assuming always that color-vision is not affected at the same time.

In the normal field the color-circles from the periphery to the centre are disposed as follows: *blue, yellow, red, green, violet.* At least such is the

order when we employ bright, saturated colors.[1] In practice it is sufficient to confine the examination to three colors,—blue, red, and green. The chief characteristic of hysterical dyschromatopsia is that the field for red is the last affected. As a result of this peculiarity the space occupied by the latter may exceed in size the field for blue.

We have with this inversion of the color-field much more certain evidence of dyschromatopsia than in its limitation alone.

As the amblyopia increases, the perception of color disappears towards the fixation-point in this order: *green, blue, red.* There are exceptions to be noted, as, for example, when to the concentric contraction of the field is added a central scotoma.

The persistence of the red field, owing to the predilection, before referred to, of hysterical subjects for that color, is the dominant characteristic of this form of dyschromatopsia. It is all the more remarkable, since in tabetic and alcoholic amblyopia the red field is the first affected.

We know that alcoholic dyschromatopsia takes the form of a scotoma that develops from the centre towards the periphery, as opposed to retinal anæsthesia, in which the limitation is concentric, and first affects red, then green, and finally violet. Indeed, contractions of the color-field in these

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[1] Ole Bull, Hess, and more recently Hegg, a pupil of Pflüger, have experimented with pigments mixed with certain proportions of black and white, for the purpose of obtaining compounds possessing the two qualities of brightness and saturation, so that the colors may have the same value in terms of white, and that they may be readily recognized at the periphery of the field in the shades proper to them.

The purpose is an excellent one, but it is a question whether it is theoretically possible to accomplish it, and whether, practically, these pigments are superior to the saturated color-paper of commerce.

We know that it is not only very difficult to obtain pigment mixtures having a fixed color-value, but that it is still more difficult to preserve the colors when once obtained; in the course of time they are prone to undergo changes. Even supposing, however, that one could obtain pigments that are unalterable and that do possess a constant white value, the problem would still remain unsolved, because their (white) tone-value varies with the character of the illumination. Bull has very justly observed that the result obtained by their employment depends upon the time of the day and the intensity of the illumination. An explanation of this fact appears in my communication (Académie des Sciences, Paris, 1884) on the luminous intensity of the spectral colors. The fact that variations in the luminous intensity of the (white) tone-value of color are influenced by the degree of the illumination, results from the increased sensibility of the retina when the illumination is diminished. It affects unequally the different refracted rays. It has no influence upon pure red, but is marked in the case of the more refrangible colors, such as blue and violet. Moreover, this increase in retinal sensibility has a certain bearing on brighter illumination; the particular color thus rendered more luminous appears less saturated, and finally passes into white. Thus it is that the saturation of a color does not depend alone upon the proportion of white which it contains, but also upon the condition of the retina.

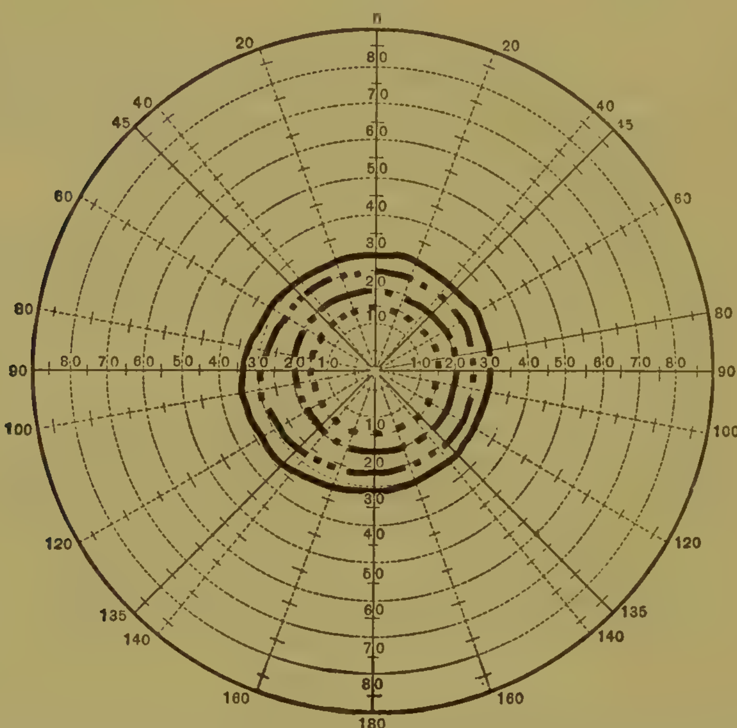
The practical value of these pigments will be determined by the curious fact that perimetric observations with them have shown that the field for red is just as large as that for green! This is certainly not the conclusion one arrives at in using spectral colors. The saturated pigments of commerce give results more in conformity with those obtained from simple rays.

diseases form two exactly opposite types, and in fact all varieties of Daltonism appear to conform to one or other of them.

Occasionally one encounters cases of ocular hysteria in which the color-scotomata present all the characteristics of toxic amblyopia. These are well shown, for example, in the case of a young girl, aged twelve, who was certainly not an alcoholic, but who was decidedly hysterical.

To the central defect is sometimes added a peripheral contraction, so that we obtain, as a result of this double perimetric limitation, an annular color-field in the shape of an intermediary zone lying between a central circle and

FIG. 2.




The visual field for red is larger than that for blue (the first symptom of hysterical dyschromatopsia).

a peripheral zone. These cases are very rare: they depend upon differences in the action of the disease.

Another exceptional symptom is not less curious. When the insensibility to light and the absolute scotoma are most marked, it not infrequently happens that the field for red may be larger than that for white. The same fact may be noticed, although more rarely, in regard to other colors. That is to say, under certain conditions colors are perceived as such, or as gray, with an intensity greater than that for white.

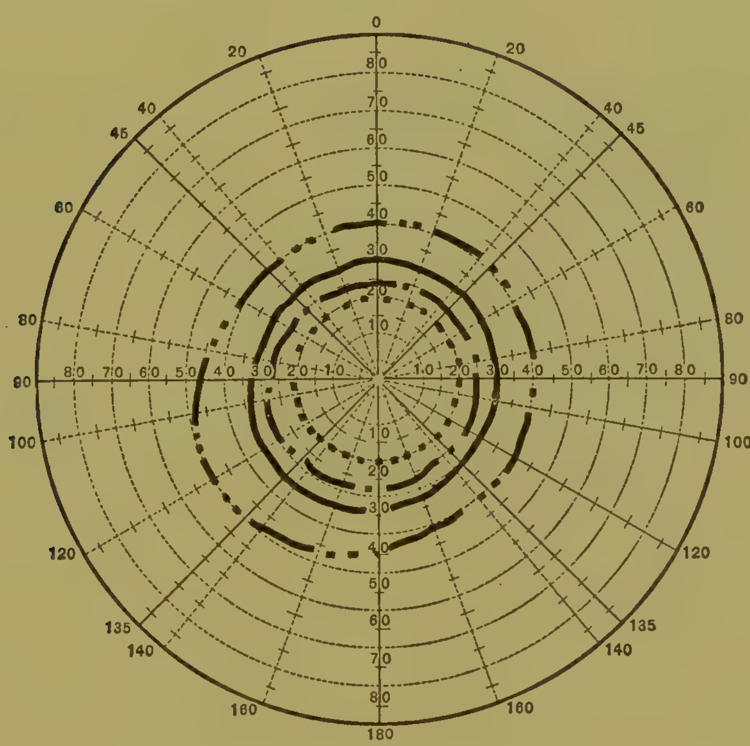
It is evident that, given the same amount and kind of light, a piece of paper whose color is produced by the absorption of a portion of the rays falling upon it represents an illuminating power less than that of white paper, which reflects almost all the incident rays.

The color produces a luminous sensation which, contrasted with the white, acts in an unexpected manner. With patients who present this peculiarity in a marked degree—who, for example, have a color-field for red almost twice as large as that for white—I have had the following experience.

The white light of the photoptometer is perceived only when of extreme intensity, perhaps a hundred times stronger than normal. When I regulate the instrument at ninety intensities the patient sees nothing. Upon interposing over the hitherto unperceived white surface a saturated red glass that absorbs almost all the rays except red, the patient immediately reports a luminous impression. I have already called the attention of physiologists to these facts, in my first memoir.

Wherever I have observed dyschromatopsia in hysterical amblyopia, it has been accompanied by a modification of the normal white-light perception. Frankl-Hochwart and Toplanski report similar observations, where, without contraction of the field for white, there was defective perception of certain colors, especially of blue.

FIG. 3.




The visual field for red is larger than that for white.

(c) *Visual Acuity.*—Perception of form is variously affected in hysterical amblyopia, but, speaking generally, it is very little interfered with, and this may be regarded as one of the distinctive features of the disease.

At first sight the acuity of vision is low, but on investigation this is usually found to be the result of accommodative anomalies whose influence as a factor in the disease it is difficult to estimate, on account of their extreme variability. The determination of distant vision is often impossible. Under ordinary conditions the *punctum proximum*, *p*, and the *punctum remotum*, *r*, tend to coalesce at a certain distance from the eye. Now, if we determine the acuity at that particular distance, exercising due care not to fatigue the patient by a too prolonged examination, we shall be surprised to find in many cases an acuity approaching the normal, although accompanied by a high degree of amblyopia, a field of vision contracted

to thirty or forty degrees, and even a notable reduction of the normal light-perception as measured by the photoptometer. It is remarkable also that, in spite of this anæsthesia for light-impressions, the visual acuity should be generally better with a moderate than with a strong illumination.

In certain cases correcting lenses improve vision to an extent that is difficult to explain by the laws of refraction alone. The writer believes that, by the intervention of the accommodative power, spherical glasses modify the sensibility of the yellow spot, just as we know they influence peripheral vision and bring about an enlargement of the visual field.

All these causes serve to produce considerable variation in the visual acuity of the same object, so that its exact determination is beset with much difficulty.

(*d*) *Monocular Diplopia and Polyopia. Micro-Megalopsia.*—Monocular diplopia is a not infrequent complication of hysterical amblyopia, but generally the patient does not definitely complain of it, apart from the defective vision; it must be sought for in some such manner as the following. A small object—a match, for example—is held in a vertical position before the eye and at such a distance from it that it is seen singly and distinctly. At a point nearer to or farther from the eye the diplopia will be made manifest. However, as the object is being moved backward and forward in front of the eye, it will be noticed that before it is seen double its outlines become indistinct in consequence of the formation of confusion-circles on the retina, just as when in ordinary fixation the eye cannot accommodate for the desired distance. The second image, more or less distinct, usually separates from the first on the temporal side.

Occasionally, as we continue the examination, a third image, still more imperfect, is perceived by the patient on the opposite side. This form of diplopia possesses one important characteristic: when it appears beyond the point of single and distinct vision it can be made to disappear by the use of appropriate concave glasses. When, on the other hand, it is seen on the proximal side of the point of single vision, the same result is brought about by the use of convex glasses. I may add that by carefully interposing a small screen in front of the pupil the images may, one by one, be made to disappear.

These peculiarities of monocular hysteria, which the writer studied[1] more than sixteen years ago, and which he has since frequently confirmed, demonstrate that, although of nervous origin, they are still due to an error of refraction, and result from physical causes. Multiple images of an object are actually projected upon the retina and are perceived as such.

It is easy to understand that when diplopia is present there must also be spasm of the accommodation. Later the writer will discuss this accommo-

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[1] De la polyopie monoculaire dans l'hystérie et les affections du système nerveux, Annales d'oculistique, May–June, 1878.

dative spasm, which almost always accompanies the amblyopia of hysteria, but which may occur without it. This condition of spasm is more or less of a complete bar to efforts at accommodation. The amplitude of accommodation is practically *nil*, and the eye is adapted for distinct and single vision at a fixed point. Either in front of this point or between it and the eye all images are double. Spherical glasses simply serve to focus the image at the point for which the eye is adapted. These phenomena are more readily determined in spasm than in paralysis of accommodation, for polyopia also accompanies paralysis of the ciliary muscle, and it can be demonstrated in normal subjects whose eyes are under the influence of atropine.

I attribute the immediate cause of this phenomenon to structural defects in the crystalline lens. The disposition of the anterior portion of the lens in three segments favors its changes in curvature, but at the same time, owing to their separation from one another by layers of amorphous material, each section possesses a focal point of its own, capable of producing images distinct from that yielded by the lens as a whole. This production of polyopia may be compared with the classic experiment of Scheiner, and, in fact, is explained in the same way. Monocular diplopia due to accommodative spasm may be produced experimentally in persons having a sufficient amplitude of accommodation. We all know that by tracing a line upon a blackboard placed at a certain distance from the eyes, and fixing a nearer object, but with the attention directed to both, the line will be seen double. This is owing to faulty convergence. In the same way, even when one eye is closed, there will be double images on account of insufficient accommodation.

Bearing these facts in mind, it is easy to understand, as was pointed out in my first memoir on this subject, that a condition of spasm is more favorable to the production of these phenomena than is paralysis, inasmuch as the former, by increasing the convexity of the anterior surface of the lens, serves to emphasize the diplopia arising from structural defects in the crystalline lens.

Such, then, are the characteristics of monocular polyopia in hysteria. They are connected, as I have shown, with conditions present in the affected eye, and result from refractive errors. Multiple images are perceived simply because they are actually projected upon the retina. Is there in hysteria, or in any other form of nervous disease, a monocular diplopia distinct from the foregoing, having its seat in the brain, while only one image is really thrown upon the percipient retina? Certain writers assert that external cerebration of this sort is possible, but I have never observed any evidence of it. It is true that there are atypical cases of monocular diplopia, where, for example, the double vision is not corrected by glasses, and where it persists at all distances from the eye. Before, however, we can admit this form of diplopia to be of purely central origin it must be shown that it is not due to irregular astigmatism (a frequent cause of polyopia) rendered

more manifest by some anomaly of accommodation. The writer must insist upon this objection all the more strongly because, in those published cases in which affections of the central nervous system are invoked to explain the multiple images, the condition of the refraction has generally received insufficient or no attention.[1]

Spasm of the accommodation, hysterical spasm especially, is the cause of another symptom, the perversion of that faculty by means of which we are enabled to estimate the true size of objects in spite of considerable variation of the size of their retinal images as they approach or recede from the eye. A pencil or a match held at various distances from a healthy eye always gives an impression of equal size. Make the same experiment in front of an eye affected by accommodative spasm, and the object appears to increase in size when it is held near and to grow smaller when it is withdrawn. In other words, the appearance of the object corresponds exactly to the size of the retinal image. It sometimes happens that patients see objects increase in size as they are withdrawn from the eye, although the retinal image necessarily grows smaller. The apparent enlargement, in these cases, seems to be due to the formation of diffusion-circles on the retina. Indeed, in monocular diplopia the single image of an object appears large owing to the juxtaposition of the two imperfect images just before they are seen to be distinctly separate. The writer has designated the increase and diminution in apparent size of an object, under the circumstances above mentioned, by the term *micro-megalopsia.* This symptom is almost a constant one in hysterical amblyopia associated with simple spasm of the accommodation. It is very useful in determining the nature of the amblyopia and in proving its existence in cases where, for instance, there is a slight contraction of the visual field that does not exceed the confines of certain normal limitations. When this symptom is the only one present, and it is not marked, too much importance should not be attached to it, because other accommodative abnormalities may produce it. We might

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[1] I except one carefully reported example from the pen of Roder (A case of hysterical diplopia, Klinische Monatsblätter für Augenheilkunde, November, 1891) which presents several unusual peculiarities. Roder dwells upon the fact that, in his case, the monocular diplopia persisted in spite of the use of atropine, and concludes that this negative result must throw out of court the claim that the double vision was due to accommodative spasm or admitted of a similar physical explanation. It must, however, be remembered that monocular diplopia is present as well with paralysis as with spasm of accommodation, and that consequently the result with atropine is an insufficient reason for admitting (as Roder does) the truth of the old hypothesis that a single retinal image may be doubled by both the crossed and the uncrossed fibres of the optic nerve. If so, how does he explain the fact that the diplopia was not produced by an object held at all distances from his patient's eye, but only, as he states, at fifty centimetres? On the contrary, it is when the object is brought nearer to the eye that its retinal image becomes larger and is most readily seen double by the various nervous elements. It is quite possible that Roder may have unwittingly practised suggestion upon his patient,—an experience which I have myself had. It is necessary, as has been previously observed, rigorously to exclude from a clinical examination every observation into which the possibility of suggestion may enter.

regard it as expressing a loss of the muscular sense, a sensory abolition commonly met with in hysterical persons, and affecting those muscular movements which ordinarily are under the control of the will.

(*e*) The Preservation of the Pupillary Reflex in Hysterical Amaurosis, and its Meaning. The Disappearance of Monocular Amaurosis in Binocular Vision. Physiological Deductions. Analogous Facts relating to Color-Vision. Relation of Hysterical Amblyopia to other Anæsthesias. Hysterical Amblyopia commonly Bilateral, but may be Monocular. No such Condition as an Hysterical Hemiopia.—The writer must again refer to certain curious peculiarities of hysterical amblyopia or amaurosis, in connection with which we are disposed to accuse the patient of simulation because they do not agree with those preconceived and more or less correct notions which we have formed of the visual apparatus.

In the most pronounced case of hysterical amaurosis the pupil reacts to light (as Mendel has pointed out), as it does not do in blindness from other causes. In other words, although the luminous impression is not perceived, it is carried to the brain in such a way as to produce the pupillary reflex. This fact is an important aid to an understanding of hysterical phenomena. It furnishes material proof of a condition which Pierre Janet[1] has shown to be not uncommon in the sensory anomalies of hysterical persons.

This fact will explain why these individuals suffer very little inconvenience from a permanent contraction of the visual field to ten or fifteen degrees,—quite a different state of affairs from what would follow a similar limitation of the field from purely ocular causes. It is probable that retinal impressions, although unperceived, may in the same way bring about in the muscular apparatus those instinctive and unconscious movements by means of which we find our way through the external world. It is rational to admit that unconscious retinal impressions may determine the performance of reflex acts by the extrinsic muscles, just as they have been shown to regulate the size of the pupil.

Several years ago the writer called attention[2] to another peculiarity of hysterical vision even more curious than the foregoing. A patient completely amaurotic in one eye, without perception of form or light, had binocular vision. Numerous and various tests made of such cases leave no room for doubting the truth of the statement. The simplest proof of this fact is gained by placing a square of paper in front of the patient. Having assured one's self that he sees the object with one eye only, let him open both eyes, and place a strong prism, axis horizontal, in front of the better eye. The patient, instead of seeing, as expected, but one image of the paper object, sees two. By varying the direction of the axis and its posi-

[1] L'anesthésie hystérique: conférences faites à la Salpêtrière, Archives de neurologie, 1892, Nos. 69 and 70.

[2] Thèse d'agrégation de Grenier: des localisations dans les maladies nerveuses, 1886. Amblyopie hystéro-traumatique: considérations sur la vision binoculaire et les amblyopies réflexes, Société d'ophtalmologie de Paris, June, 1889.

tion, by placing the base up or down, and by covering the blind eye during the act of binocular fixation, one can be certain that one of the images belongs to the eye which in monocular fixation is absolutely amaurotic. These control experiments can also be made with the stereoscope or with the instrument of Flees.

The recovery of vision in the amblyopic eye at the moment of binocular fixation affects central vision only; peripheral sight remains abolished both in binocular and in monocular vision, because, so far as the affected eye is concerned, perception does not extend to more than eight or ten degrees from the fixation-point. The acuity of central vision, other things being equal, is as perfect in the amblyopic as in the healthy eye.

In some of these cases the unilateral amaurosis does not disappear during binocular fixation, either because the defective sight is too marked in the affected organ, or because the individual never possessed binocular vision or had it in a very imperfect degree. Hysterical amblyopes who present binocular vision, on the other hand, may be divided into two classes, —those who always have it, and those who need some such aid or artifice as prisms or the stereoscope to call it into being.

The disappearance of monocular amaurosis during binocular fixation has been observed by Schweigger (according to an oral communication of Samelsohn), by Bernheim,[1] by Pitres,[2] and by Janet.[3] Bernheim offers a purely psychological explanation of the occurrence. "These phenomena," says he, "are due to a spiritual illusion; . . . it is a blindness of the imagination; . . . it is due to the destruction of the image by a psychic agent." This is language the writer does not understand. When we more thoroughly comprehend the functions of the visual apparatus these phenomena will be explained, just as other affections of the organs of sense are explicable by studying their physiology.

For the interpretation of hysterical visual symptoms and other pathological verities, such, for instance, as the different forms of transitory amblyopia or some peculiarities of vision in squint, the writer long ago[4] predicated the existence of certain relations between the retina and the visual centres in monocular and binocular sight, as well as definite and distinct relations in central vision and in peripheral vision. Frankl-Hochwart,[5] studying the characteristics of vision in hysteria, has arrived at identical conclusions. It is true that, since the days of Graefe, lesion of one hemisphere is admitted to produce only hemiopia, and Mauthner has not neglected to

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[1] De l'amaurose hystérique, Revue de l'hypnotisme expérimentale et thérapeutique, 1886.

[2] Leçons cliniques sur l'hystérie, Paris, 1881.

[3] L'anesthésie hystérique, Archives de neurologie, 1892.

[4] Des rapports croisés et directs des nerfs optiques avec les hémisphères cérébraux, Société de biologie de Paris, March 11, 1882, and Bulletins de la Société française d'ophtalmologie, 1884, p.52. Amblyopie hystéro-traumatique : considérations sur la vision binoculaire, Société d'ophtalmologie de Paris, June, 1889.

[5] Des anesthésies cérébrales, Société impériale des médecins, Vienne, February, 1893.

oppose this objection to Frankl-Hochwart's views; but one can say, with Charcot, that questions involving hysterical phenomena are not to be settled by anatomical investigations, but by physiological laws. There is no way in which we can readily understand the function-processes of the visual apparatus unless we conceive of functional relations between the two hemispheres by way of commissural fibres in the corpus callosum. These connections are not entirely hypothetical. In a case of long-standing hemiopia produced by a lesion of the cortical sight-centre, observed by Dejerine, he found a degeneration of the fibres in the posterior portion of the corpus callosum. Further, Mott[1] has demonstrated the association of the motor centres in the two hemispheres.

It is almost certain that hemianæsthesia is the result of a functional disturbance of the opposite hemisphere. If we admit the crossed relation between each eye and the opposite hemisphere in monocular vision, the left eye will become amaurotic when the right side of the brain is affected; but if in binocular vision both eyes are connected with a single hemisphere, it seems clear that if that hemisphere be the left and healthier one, the left and amaurotic eye will be able to see. It is in this way that the writer explains an observation similar in character made by Charcot and Féré relative to color-vision.[2] Suppose a patient color-blind for green, which he sees as gray in his left, amblyopic eye. Placing before the healthy eye a prism that produces double images of a green paper object, it will be found that the patient does not see, as one might expect, a green and a gray image, but two green images; and if the eye be not too amblyopic, the prism before the affected eye may occasion two gray images. This experiment serves to illustrate my contention. The two eyes having been connected with the diseased hemisphere, the eye that sees normally in monocular fixation becomes amblyopic in binocular fixation.

The writer has also observed patients color-blind in each eye separately who could distinguish colors quite readily with both eyes.

Regnard and Grasset have shown that a person blind for a certain color sees as white a mixture of the color with its complementary when they are both placed upon Newton's disk. In reporting these experiments it should always be stated whether monocular or binocular vision is referred to.

While investigating the effects of color-contrasts upon hysterical patients, the writer found that the sensation induced by a color that is not perceived can be developed, provided the complementary color inducing it is seen normally. The converse of this is, however, not true: we cannot develop the sensation proper to a certain color, although it may be perceived by the objective excitation when there is blindness for the complementary color.

The dominant characteristic of hysteria is the production in the sensory

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[1] British Medical Journal, May, 1890.

[2] Bulletins de la Société française d'ophtalmologie, 1884, p. 52.

apparatus of disorders as numerous as they are varied. The skin, the mucosæ, all the organs of special sense, singly or simultaneously, may be affected, and most commonly under the guise of a hemianæsthesia. Hysterical amblyopia may be styled an anæsthesia of the organ of vision. According to Dana,[1] it is the most frequent of all the hysterical anæsthesiæ, and, thanks to the precise methods of examination possessed by us, is the easiest of recognition. It is also of extreme importance on account of its clearly defined characteristics and the precise nature of the information which it furnishes.

Hysterical amblyopia is almost always accompanied by other disorders of sensation, especially of the pharynx, skin, and mucous membranes. It is probable that when it appears to be the sole sensory symptom present, it is so because evidence of other anæsthesiæ is not so easily elicited. In typical cases of hysterical amblyopia we find more or less complete hemianæsthesia of the skin, of the mucosæ, and of the other sense-organs.

In other instances the anæsthesiæ are more or less localized, and among these may be mentioned cases where the loss of sensation is limited to the skin about the amblyopic eye. These phenomena, studied by Charcot and Féré, are not uncommon. Not only may the palpebral skin become quite anæsthetic, but the conjunctiva and the cornea may also be absolutely insensitive. This anæsthesia of the conjunctiva—of the left especially—occurs so frequently in hysteria that Briquet regards it as characteristic of that condition. As Gilles de la Tourette points out, the entire cornea rarely loses its sensitiveness; the inner or outer half may be insensitive, but the central portion is commonly normal. According to Féré,[2] when the anæsthesia of the conjunctiva and cornea is complete, the oculo-palpebral reflex is defective. If, while an object is carefully fixed, a roll of paper is brought in contact with the conjunctiva and cornea, the lids remain motionless as long as the pupillary area is not touched. The reflex is excited, not by irritation of the conjunctiva or cornea, but by the effect upon the retina.

It is always to be remembered that, notwithstanding the entire absence of sensation in the conjunctiva and cornea, and in spite of the abolition of the oculo-palpebral reflex, the lid-movements are normally executed, contrary to what occurs in trigeminus lesions, for example. The lacrymal reflex, according to Gilles de la Tourette,[3] is not interfered with, even when the oculo-palpebral reflex is abolished. "Whenever," says he, "we found the conjunctiva insensitive to the touch of a piece of paper, the tears were secreted immediately and as abundantly as when the mucosa preserved its normal sensibility." Pitres also noticed the conservation of the glandular reflex in the conjunctival anæsthesia of hysteria.

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[1] A Study of the Anæsthesia of Hysteria, American Journal of the Medical Sciences, October, 1890.

[2] Note pour servir à l'hystérie de l'hystéro-épilepsie, Archives de neurologie, 1882.

[3] De la superposition des troubles de la sensibilité et des spasmes des muscles de la face, Nouvelle iconographie de la Salpêtrière, 1889.

Although most commonly associated with hemianæsthesia, hysterical amblyopia usually affects both eyes.

A careful study of the cutaneous anæsthesia of hysteria shows that it is more rarely unilateral than is generally supposed. I have long since suspected, with Ballet,[1] that in cases of bilateral amblyopia the skin on both sides of the body is also affected, and that the hemianæsthesia merely expresses a greater degree of insensibility on one side than on the other.

When the amblyopia affects both eyes, it is usually more pronounced on the side that shows the more marked amount of other sensory disturbances. This rule is, however, not an invariable one: sometimes the opposite eye is most affected.

Undoubtedly there are cases of pure hemianæsthesia in which the insensibility seems to be strictly limited to one-half of the body. A true unilateral, hysterical amblyopia, at one time regarded as of doubtful authenticity, is not extremely rare. Of eighty-eight cases occurring in my service at the Salpêtrière during the year 1888, M. Morax,[2] my assistant, found eight in which the amblyopia or amaurosis was confined to one eye. There were even cases in which, with a complete unilateral amblyopia or amaurosis, the field for white and colors was abnormally large in the unaffected eye. This corresponds to the cutaneous hyperæsthesia noted by such observers as Freund, Frankl-Hochwart, and Topolanski.

When there is a transference of the anæsthesia, the monolateral amblyopia is displaced in the same manner as the other sensory symptoms.

I have insisted upon the fact of a unilateral amblyopia because it entirely disproves such hypotheses as that of Wilbrand,[3] who, in order to explain hysterical amblyopia by the well-known connection of the retinal with the visual cortical centres producing the amblyopia, postulates a double hemiopia, thus rendering necessary an affection of both eyes.

Hysteria simulates even organic lesions. It may assume all possible forms of sensory anomalies. There is, however, one variety of visual insensibility that it appears to be incapable of producing, and that is hemiopia.

Nevertheless, hysterical hemiopia has been described by some authors, among them Rosenthal, Sturge, Galezowski, and Westphal. Rosenthal, in a letter to Charcot, affirmed his original opinion[4] that, thus far, it has not been shown that hysterical patients exhibit anything more than an amblyopia or a contraction of the visual field.

We must distinguish the transitory from the permanent form of hemiopia. Babinsky[5] has stated that the first form—that is to say, a

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[1] Recherches anatomiques et cliniques sur le faisceau sensitif, 1881.

[2] Compte-rendu du service ophtalmologique de la Salpêtrière pour 1888, Archives de Neurologie, 1889

[3] Klinische Monatsblätter für Augenheilkunde, 1885.

[4] Gilles de la Tourette, loc. cit., p. 878.

[5] Babinsky, De la migraine ophtalmique hystérique, Archives de neurologie, November, 1890. See also Fink, Des rapports de la migraine ophtalmique hystérique avec l'hystérie.

transitory hemiopia presenting all the characters of ophthalmic migraine or scotoma scintillans—is sometimes attributed to hysteria. It may appear alone or as the aura of a convulsive attack. It can also be provoked by suggestion. I examined one of the patients (whose case was studied and reported by Babinsky) during an attack of ophthalmic migraine produced by suggestion. She said that she saw only one-half of objects, but I could not discover, after a perimetric examination, anything more than an exaggeration of a concentric contraction that was present before the attack.

As to the permanent form of hemiopia, presenting the usual signs of central lesions, I have never seen it in hysteria, except as coincident with the organic alterations referred to. To this statement I may add that in the Salpêtrière neither Charcot nor his pupils ever found a single example of hysterical hemiopia among the thousands of patients examined by them. I believe that published statements to the contrary are erroneous. This is also the opinion of Gilles de la Tourette.[1] Finally, Freund[2] agrees that hysteria never produces hemiopia.

Hysterical amblyopia being entirely a functional disease, it is easy to understand that it is not accompanied by morbid alterations in the background of the eye. If such exist, the writer has never seen them. It is true that certain authors—Landolt in particular—speak of enlargement of the retinal vessels, of serous exudations into the substance of the retina, and, in one case, even of a partial atrophy of the optic nerve.

"One very often finds," says Nuel,[3] "more or less hyperæmia of the papilla and sometimes of the retina. In time the disk becomes slightly clouded, obscures the lamina cribrosa, becomes discolored, and assumes a grayish tint. In cases where the amblyopia is considerable and the perimetric contraction of long standing, the gray discoloration of the papilla finally merges into a simple atrophy. True neuritis has also been observed, but in such instances one may well inquire whether it is not due to some other morbid agency."

Not only in cases of optic neuritis, but in all the other organic fundus-lesions of hysterical patients, it would be well to ask the same question.

## AFFECTIONS OF THE MOTOR APPARATUS.

(*a*) *Disorders of Accommodation.*—Of all hysterical affections of the eye, and of hysterical affections in general, those of the accommodation are the most frequent. They almost always accompany the amblyopia, and they often exist without it. It is not rare, in children, to find them as the earliest manifestations of the neurosis,—a manifestation which does not always imply a very grave prognosis.

It was not possible that such a common symptom should long remain unrecognized. It has been described under various names: the *painful*

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[1] Loco citato, p. 373.

[2] Club médical de Vienne, May 24, 1893.

[3] Amblyopie hystérique, Traité complet, vol. iii. p. 720.

*accommodation* of Donders, the *hysterical kopiopia* of Förster, the *hyperæsthesia of the ciliary muscle* of Nagel, which all refer to the same affection, —*i.e.*, to modifications of the accommodation that seem to be always of hysterical origin, even when the neurosis does not manifest itself by the other symptoms.

These anomalies of the accommodation are essentially of the nature of spasm. However, the most frequent symptom, or at least the one which appears most urgent in the milder forms of the disease, is insufficiency of accommodation, simulating an incomplete paralysis. Steffan[1] thinks that paralysis and spasm occur with equal frequency. Chibret[2] has quite recently acknowledged that the accommodative anomalies which he observed so frequently in children are hysterical in character, but he calls them a paralysis of accommodation.

In 1878 the writer investigated the subject of hysterical spasm of the accommodation in connection with monocular polyopia, first in his article on anæsthesia of the retina,[3] and afterwards in a communication on paralysis and contracture of convergence.

The point that characterizes paralysis of the accommodation, such as one observes in connection with third-nerve palsy or that artificially produced by atropine, is that the *punctum proximum*, *p*, approaches the *punctum remotum*, *r*, and is coincident with it when the paralysis is complete. Spasm of the accommodation, such as one produces by eserine, identical with that observed in marked cases of hysterical spasm, is signalized by the displacement of *r* towards *p*, and its final identification with it. When one studies the evolution of hysterical spasm of the accommodation, it is seen that its chief characteristic is the displacement towards each other of *p* and *r*. In other words, the amplitude of accommodation is diminished at both ends of the line. In consequence of the recession of *p*, the eye acts as if it were presbyopic or as if affected by paresis of accommodation. As a result of the advance of *r*, there is at the same time a spasmodic myopia. In this way convex glasses relieve the visual defects attendant upon accommodating for near work, while concave lenses relieve distant troubles.

As long as *p* and *r* do not coincide, the patient enjoys a certain amount of amplitude of accommodation, but when fusion is complete the amplitude is nil, and the person is able to see distinctly at a fixed point only.

The point, moreover, where fusion takes place is variable. It may be more or less remote from *p* or from *r* without losing the characteristics of spasm, just as a limb affected by a contracture may assume the position of complete flexion, of moderate flexion, or even of extension. It is con-

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[1] De l'anesthésie de la rétine avec rétrécissement concentrique du champ visuel, Bericht der Ophthalmologischen Gesellschaft, Heidelberg, 1873.

[2] Loco citato, 1892.

[3] De la polyopie monoculaire dans l'hystérie, Annales d'oculistique, 1878. De l'anesthésie de la rétine, Annales d'oculistique, 1886. Paralysie et contracture de la convergence, Société d'ophtalmologie de Paris, 1889, p. 171.

sequently easy to understand how the distance of the fusion-point from *p* and *r*, with reference to the eye, will be influenced by the myopia, hypermetropia, or presbyopia in the case.

Bearing these facts in mind, one may readily see why an hysterical paralysis of the accommodation, if it does exist, must be extremely rare. Continued observation of the same patients will bring out the fact that an apparent paresis of the accommodation in children may in a day become converted into a case of marked spasm.

I have already cited the case of a child, recently under my care for nervous asthenopia with paresis of accommodation, who developed within twenty-four hours a spasmodic myopia of eight diopters, accompanied by contraction of the field of vision.

Hysterical spasm is easy of recognition. To begin with, there is the evidence of test-lenses: only one must remember that the result of the examination may vary with the amount of spasm. When it is marked, one sees the very high degrees of spasmodic myopia noted by Steffan and Galezowski.[1] This myopic spasm is, I believe, always hysterical in character, and it is habitually accompanied by contraction of the visual field. It is easily distinguished from axial myopia by an examination with the erect image or by retinoscopy, and it disappears under the influence of atropine.

Another type of the affection, more frequently observed, expresses a minor degree of accommodative weakness, and shows the usual symptoms of insufficient accommodation by the recession of *p*. When a child, evidently emmetropic, is obliged to hold fine print at a distance of fifteen to twenty centimetres to see it distinctly, and when a +1 or +1.50 D. lens decidedly improves near vision, one may feel pretty certain, if diphtheria can be excluded, that the patient is affected by a mild form of hysterical contraction of the accommodation.

In a third category of cases (probably the most characteristic of all) there is spasmodic myopia in conjunction with an insufficiency of the accommodation for near vision. In establishing these symptoms the practitioner will also search for the micro-megalopsia that always exists, and the peculiar monocular polyopia described by me that frequently accompanies it.

Before leaving this subject, a few words must be said concerning pupillary anomalies: these occur very rarely as permanent manifestations of ocular hysteria.

Contraction and dilatation of the pupils have no precise significance in nervous pathology, except as expressing a reflex act. In hysterical subjects we find the pupils sometimes contracted (especially when photophobia is present) and sometimes dilated, but with the reflexes preserved and without pupillary inequality. In young neuropaths one occasionally notices a slight pupillary inequality, particularly in the presence of a weak illumination, but without any appreciable anomaly.

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[1] Le progrès médical, 1878, p. 39.

I have seen this pupillary inequality especially among epileptics. I have not noticed the myosis that, according to Galezowski, accompanies spasmodic myopia.

Mydriasis is described as occurring in several instances reported by Harlan,[1] Duboys,[2] and the writer.[3] Of the last case a *résumé* is given on another page.

There are reservations to be made in speaking of hysterical mydriasis. It is wise to remember, in cases that resemble one another so closely, the possible coincidence of two diseases. A monocular mydriasis, with abolition of the reflexes, may follow trauma of the face or irritation of branches of the trigeminus. The writer would not assert that the presence of hysteria is necessary to explain these reflex mydriases.

(*b*) *Anomalies of Convergence.*—Innervation of convergence and weakness of accommodation are often associated in hysteria. In the minor degrees of these two affections, however, convergent anomalies attract much less attention than accommodative insufficiency, because, possibly, they are not so easily demonstrated, and because they cause the patient less annoyance.

The function of convergence is affected in various ways, and we shall find in these variations an excellent example of the disassociation of the constituent elements of a function, thus presenting one of the most remarkable characteristics of hysterical disorders.

In the normal condition there are two principal factors in the production of convergence,—accommodation and fusion. In hysteria these factors may be affected either separately or simultaneously.

In the first class of cases the muscular asthenope always complains of more or less clearly marked symptoms of the asthenopia. With a prism, base horizontal, which disassociates the two images we find insufficiency of convergence. We verify this insufficiency by causing the patient to fix a near object, covering each eye alternately. On examining the state of distant convergence with a prism having a horizontal base over one eye and a colored glass over the other, the patient looking at the flame of a candle, we may find defective, normal, or excessive convergence. In the last variety, which shows that the disorder in general is more pronounced than in the preceding varieties, the far point of convergence is brought nearer the patient, while the near point is farther removed,—that is to say, defect of divergence for distant fixation and defect of convergence for near fixation may coexist, convergence being disturbed at both extremities of its range.

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[1] Case of Blindness with Violent Blepharospasm and Mydriasis, Transactions of the American Ophthalmological Society, 1884.

[2] Mydriase double de nature hystérique, Bulletin de la Clinique nationale ophtalmologique de l'hospice des Quinze-Vingts, 1883.

[3] Paralysie des mouvements associés, Archives de neurologie, March, 1883. Paralysie et contracture de la convergence, Bulletin de la Société d'ophtalmologie de Paris, 1889, p. 170.

In a word, we find an anomaly of convergence which presents the same characteristics as a disorder of accommodation. Disturbances of accommodation almost always accompany those of convergence.

Although these patients do not complain of diplopia during fixation upon objects at different distances, we are yet able to discover, by the methods of examination already described, a lessened convergence, or even an absence of convergence. If, however, the convergence is examined simply by making them fix a finger, gradually brought nearer the eyes, these patients are occasionally capable of excessive convergence.

How is it that we find this difference in the amplitude of convergence after using these methods of examination? Why, also, do not these patients complain of diplopia? It is because convergence is affected in only one of its phases. The accommodation-convergence function alone is deranged; convergence continues to be active because of the necessity for fusion. This is why, in order to recognize the anomaly of convergence, we are obliged to have recourse to the classic use of a prism with horizontal base, which overcomes efforts at fusion, or to occlusion of each eye alternately, which produces the same result.

In another class of cases, more rare, but very characteristic, the two factors of convergence are simultaneously affected. It is in such instances no longer necessary to resort to the artifices just mentioned in order to recognize the anomaly of convergence. It is sufficient to put a red glass over one eye and to make the patient fix the flame of a candle, or, better, a luminous slit. The patient will at once complain of diplopia. We can then easily study the anomaly of convergence, which presents the same features as those of the preceding class. Insufficiency is often the dominant sign, the symptoms being those of a more or less complete paralysis of convergence; but there is habitually a defect of divergence for distant fixation, and at the same time a defect of convergence for near fixation. In the most typical cases there is single vision at a point about two metres from the eyes. On the proximal side of this point there is crossed diplopia; on the distal side it becomes homonymous. This diplopia, whether homonymous or crossed, presents the characteristic that separation of the images is not perceptibly increased by lateral movements to the left and right, which shows that it is not due to a muscular error, but concerns simply movements of convergence.

In certain cases fusion is effected at a distance of about two metres. With other patients it is impossible to obtain fusion, notwithstanding the equal and sometimes normal visual acuity of both eyes. When the luminous slit is removed, the crossed images suddenly become homonymous, without transition. These cases belong to a class that may be described as having "a horror of binocular vision," and they prove the complete absence of a cerebral centre for the fusion of images.

Accommodation is modified in much the same way as convergence, but there is no absolute correlation between the extent of the nervous disturb-

ance of accommodation and that of convergence. Extreme degrees of spasm of accommodation are not, as a rule, accompanied by an equal amount of contracture of convergence, although very high degrees of contracture of convergence may be present in strabismus. In a case of Ulrich's, which I shall describe, an hysterical convergent strabismus possessed practically all the characteristics of contracture of convergence.

In my first book, published in 1883, I described the preceding phenomena under the name of paralysis of convergence and divergence. Later I regarded them as signs of contracture.

In typical cases of contracture of convergence exhibiting spontaneous diplopia we again find, so far as concerns the state of convergence when tested by the fixation of a finger slowly brought nearer the eyes, two classes of patients. In some of these patients there is no movement of convergence, or the act is imperfectly executed. In others, on the contrary, a very powerful convergence is noticed, in spite of the fact that a considerable defect of convergence may be observed. Those patients in whom convergence, with its two principal factors, accommodation and fusion, is effected simultaneously, and who present an asthenopia that renders all work impossible, may be still able to converge, even to an excessive degree, either by voluntary effort or by the fixation of an object. These phenomena cannot be explained by the distinction made between sustained and temporary efforts at convergence. Thus we have a new method of separating the factors of convergence. To understand these factors it must be understood that accommodation and fusion are not the only influences in the production of convergence. There is, for example, voluntary effort at convergence, whereby with a little practice even a more or less pronounced convergent strabismus may be developed. This voluntary effort at convergence, however, does not interfere, or at least does not intervene directly, in the performance of the normal function. Even during voluntary convergence which has not a visual act for its object, it is certain that convergence is accomplished under an influence other than that of accommodation or fusion. This third factor has been called the "distance-sense" by Hansen Grut, and the "sense of convergence" by Alfred von Graefe. This is hardly the place to inquire into the question. I merely wish to call attention to the existence of this third factor in convergence (necessary for the comprehension of pathological phenomena), and to remark that such pathological phenomena are additional proofs of its existence.

Hysterical convergent strabismus, upon which Manz,[1] Schweigger,[2] and Ulrich[3] have given excellent reports, involves, it seems to me, a high degree of contracture of convergence, all the factors of the latter being affected. I find proof of this in the peculiar character of the diplopia described by Ulrich. "The images of the homonymous diplopia," he

[1] Berliner klinische Wochenschrift, January, 1880.

[2] Klinische Untersuchungen ueber das Schielen, Berlin, 1881.

[3] Klinische Monatsblätter für Augenheilkunde, July, 1882.

reports, "have the same separation on looking to the right as when the patient looks to the left." Schweigger says, moreover, that this hysterical strabismus is characterized by a limitation in the motility of the external recti, combined with a contraction of the interni. In these extreme degrees of contracture which cause convergent strabismus the lateral excursion does not always maintain its normal extent as in ordinary cases of contracture without strabismus. This, however, is not sufficient, it seems to me, to exclude an anomaly of convergence-innervation and to substitute for it a mere muscular disturbance. We know that a contracture of convergence sufficient to develop a more or less constant strabismus may affect lateral movements as well as those presided over by a different nervous influence. Besides, as I shall show, in the extreme degrees of hysterical ocular contracture the associated movements of direction are often affected simultaneously with the movement of convergence.

Hysterical convergent strabismus from contracture seems to be always transitory, the attacks recurring by crises. It may be succeeded by conjugate deviation, as in Manz's case. When the strabismus is permanently established and presents the characteristics of concomitant strabismus, another influence should be suspected. The nervous disturbance, whose real cause lies in the presence of hypermetropia or some other condition, then occasionally results in latent strabismus.

(*c*) *Hysterical Asthenopia.*—Hysterical asthenopia is almost always due to disorders of accommodation and of convergence, as has been just described. Another factor in this variety of asthenopia is photophobia, which exists in a more or less marked degree in retinal anæsthesia. Difficulties in performing ocular work are not uncommon with hysterical patients who do not present spasm of accommodation or of convergence, but this asthenopia is not characteristic of hysteria. The "painful accommodation" of Donders, the "hyperæsthesia" of Nagel, the "hysterical kopiopia" of Förster, are all varieties of hysterical asthenopia. To this category we may also add the "hysterical ocular pain" of Schenkl. The pain may, however, exist independently of the ocular effort.

In the variety most frequently encountered, which is that observed among children, and in which accommodative difficulties predominate, the asthenopia manifests itself by obscurity of vision, accompanied by headache, with tingling and burning sensations of the eyes. The patients may complain of micropsia.

This form of asthenopia somewhat resembles that accompanying hypermetropia, inasmuch as children often consider it necessary to hold the book at a distance while reading. It differs from it in the rapidity with which fatigue is manifested. It is not produced, as in hypermetropia, after several hours of work, but often shows itself in a few minutes. Nevertheless, some children are not greatly incommoded by their accommodation difficulty, and complain of fatigue only in the evening when doing near work with artificial light. In some cases the sensation of tingling and burning

is so pronounced that it might be thought to be due to conjunctivitis. A systematic examination, such as has been described in the consideration of spasm of accommodation, will dispel these doubts.

Anomalies of accommodation often exist without appreciable effect upon the convergence, but, as a rule, convergence and accommodation are simultaneously involved. With symptoms of accommodative asthenopia are associated those which are commonly and incorrectly ascribed to muscular asthenopia, but which the writer terms the asthenopia of convergence. While reading, the letters appear to be displaced and doubled, and to the pains in the head is added a peculiar sensation of dragging. When there is abnormal convergence-innervation a condition of vertigo often appears. The writer has seen some patients in whom this symptom assumed the proportions of an agoraphobia, which was at once relieved by prisms. This vertigo is frequently accompanied by nausea. In this variety not only reading and near fixation, but also distant fixation, are painful.

In other cases pain is the prominent symptom. The use of the eyes occasions ocular and periocular pain, which, as a rule, has its seat above the orbit or at the base of the eyebrows. Upon pressure, however, we do not find painful points as in trigeminal neuralgia. If the patient continues to work, the pain assumes an extraordinary intensity, radiating over the head and occasionally extending to the neck and shoulders. This form is often accompanied by a marked degree of spasm of accommodation. The pain, which is especially provoked by use of the eyes, may also be developed outside of eye-work, and is exaggerated by various influences, such as the emotions, insomnia, etc.

Finally, the patient may complain of an intense sensitiveness to light; but, although photophobia is the dominant symptom, pain is also more or less common. Sometimes solar light, or, to be more exact, diffused but intense daylight, is painful; even artificial light may not be well borne.

Some patients are so incommoded by this photophobia that they are forced to remain in total darkness. It is almost always accompanied by a marked degree of spasm of accommodation, and it is not uncommon to see the contracture extend to the orbicularis palpebrarum and to the extrinsic muscles of the eye.

Some patients complain of special abhorrence of certain colors,—red, for example. This chromatophobia, which is distinct from photophobia proper, is not peculiar to hysteria.

(*d*) *Anomalies of the Ocular Excursions or Movements of Direction.*—In the normal condition the ocular movements are always associated,—that is, they are executed simultaneously by both eyes. These associated movements are of two kinds. The first have for their object fixation at different distances. These are the associated movements of convergence, the disturbances of which in hysteria I have just discussed. The others have for their function fixation in different directions, and these I call associated movements of direction. We have seen that in the anomalies of con-

vergence no muscular disorder is involved. The so-called insufficiency of the internal recti, which is incessantly made to do duty, is no more present in hysteria than in other diseases. It has to do with an anomaly of innervation governing a special function, and we have seen that this anomaly may occur in various forms. If we wish to understand these anomalies of the general movements of the eyes or of the movements of direction in hysteria, we must look at them both from the same point of view, and consider that neither muscles nor nerves, but nerve-centres, and, indeed, the higher centres, are affected,—those whereby the movements themselves are brought into unison with psychic action.

Hysterical disorders are referable almost exclusively to varieties of these two species of associated movements,—those of convergence and those of direction. There are, however, exceptions to this rule: in contractures due to blepharospasm, for instance, the motor disturbance may be limited to or may at least predominate in one eye.

Another fact connected with a study of hysterical disorders of the ocular apparatus is that they are almost always of the nature of contractures, even when they present the objective characteristics of paralysis. The writer has become convinced of this from observation of anomalies of accommodation and convergence. The same is true of those disorders that concern the general movements of the eyes and of the eyelids. For a long time Charcot did not believe in the existence of hysterical paralysis affecting the muscles of the head. He was obliged to modify this belief in consequence of a report made by his pupils Ballet and Babinsky on facial paralysis. There are ocular disturbances, also, which it does not seem proper to class among contractures, and which possess all the objective characteristics of paralysis, with this exception, that these paralyses are of a particular kind, and are distinguishable from organic palsy by special features.

These general considerations, in the present state of the question, seem to us more useful than the accumulation of incomplete and contradictory observations. They make it unnecessary to enter into long descriptive details of the anomalies of the movements of direction of the eyes, which are, moreover, much rarer and consequently much less important than those of convergence.

We find in a very exhaustive work by Borel[1] several cases relating to this part of our subject. These observations, however, do not have, in general, the significance which is attributed to them. The question is, therefore, very confused, and ought to be studied anew, considering the phenomena from the stand-point of our actual knowledge of hysteria.

*Hysterical Ophthalmoplegia. Associated Paralysis.*—I have for a long time pointed out in my lectures at the Salpêtrière a form of hysterical ophthalmoplegia which affects voluntary movements, the reflex and involuntary

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[1] Affections hystériques des muscles de l'œil, Archives d'ophtalmologie, vol. vi., 1886.

movements alone continuing to be executed. When the patient is ordered to fix an object which is moved in different directions, or is told to look up, down, to the left, or to the right, he finds it impossible to execute these movements, although the eyes may be moved instinctively under the influence of various excitations that bring about ocular excursions without the direct intervention of the will.

When I first spoke[1] to my colleagues in Paris of anomalies of this nature, some of them were naturally very much astonished. I even discovered sceptics among the physicians at the Salpêtrière. Now that we are better acquainted with the nature of hysterical disorders, we consider these phenomena a matter of course; indeed, dissociations of this kind are the very essence of hysteria. We find similar modifications of movement in the muscular tremor ("astasia-abasia") first described by Richer and Blocq, and about which to-day every one knows. As regards facial paralysis, Ballet has reported a case in which the paralysis was manifested only when the patient attempted to speak. Babinsky[2] has made an excellent analysis of these curious dissociations of movement, under the name of "systematic hysterical paralysis." Hysterical ophthalmoplegia affecting only voluntary movements has, since my report, been studied by Ballet and by Raymond and Koenig[3] upon a patient who was exhibited to the Medical Society of the Paris Hospitals.

This dissociation is of the same nature as that which I described in speaking of anomalies of convergence. Although these hysterical disorders are not, as a rule, ascribed to cerebral localizations, they have, nevertheless, as I have often stated, their anatomical cause. The anatomical reason for this dissociation of ocular movements has been furnished by the experiments of Munk, Horsley, Mott, and Schaeffer, who have established two distinct cortical centres for movements of the eye: one situated in the occipital lobe, superimposed, as it were, upon the visual centre and apparently controlling reflex movements; the other, in the frontal lobe, especially governing voluntary movements.

This form of ophthalmoplegia is not accompanied by diplopia, and does not appear to cause the patients any inconvenience. The dissociation that characterizes it is not always so well defined. As in anomalies of convergence where movement is affected in one of its phases, then in two, and finally in all, so here may associated movement of direction be affected. The paralysis may completely involve this movement in all its forms. I have not, however, observed paralysis of all the ocular movements in hysteria.

I have observed this complete paralysis under the form of associated paralysis. In this case diplopia is possible, for the motor trouble does not

[1] Société d'ophtalmologie de Paris, October, 1877.

[2] Société médicale des hôpitaux de Paris, October–November, 1892.

[3] Sur la dissociation des mouvements oculaires chez les hystériques, Annales d'oculistique, vol. cvi. p. 5.

necessarily involve the associated muscles of both eyes to the same extent, any more than it does in associated nuclear paralysis.

When there is a defect in associated movement, it is not always easy to determine whether it is due to contracture or to paralysis. We can, however, hardly conceive of a defect of movement due to contracture of opposing muscles without having conjugate deviation. Still, there are cases in which conjugate deviation is wanting.

*The Contractures. Spasmodic Conjugate Deviation, its Alternation and Combination with Contracture of Convergence. Monocular Contractures.*— Spasmodic conjugate deviation of the eyes frequently occurs during hysterical convulsive attacks, but the condition is rarely a permanent stigma. The deviation may be in all directions, but it seems to prefer the inferior and oblique positions. In a case reported by Frost[1] occurs the following: "Both eyes were directed downward and to the right, and almost incapable of movement. Upward movements were in particular impossible. During the examination, if the patient's upper lids were unexpectedly touched with the point of a pen, both eyes would be immediately raised in an entirely natural manner." This is an example of the dissociation of reflex and voluntary movements.

I also find in this report, "The left eye being covered, the right eye could fix perfectly and follow the finger freely in all directions." I have observed this peculiarity in certain patients, among others in a young Russian girl, who remained for a long time under the care of Charcot and was affected by blepharospasm and a left facial hemispasm. When a bandage was placed over her left eye, her right eye was able to fix in a normal manner, and, as soon as it was removed, permanent convulsive deviation occurred.

I also notice in Frost's article that the ocular deviation disappeared under the action of chloroform. In the case of this same patient the deviation persisted for two years, during which she was under observation. Conjugate deviation from contracture is generally less persistent; it is sometimes periodic, depending upon the influence of the general health, and especially of the convulsive attacks. It may change in character from one attack to another. For example, it may be replaced by contracture of convergence, as in a case reported by Manz. Finally, contracture of two varieties of associated movements, the lateral and those of convergence, may coexist. In that case there would be convergent strabismus, with defective movements in the lateral excursions of the eyes. A typical example of this variety is furnished by No. 67 in my case-records of the Salpêtrière for 1888, by my assistant, M. Morax.[2] I shall recur to this combination in discussing a certain variety of paresis which it ordinarily simulates.

In another form of contracture both eyes execute incessant movements,

[1] British Medical Journal, 1884. See also Borel's monograph.

[2] Archives de neurologie, 1889.

instead of being permanently deviated, and seem to have lost the power of fixation. These movements are more or less incoherent, and are usually accompanied by marked photophobia and blepharospasm. A case which I recently observed exhibited contracture of the levator palpebræ, and simulated Graefe's symptom in Basedow's disease, except that intermittent retraction of the lids occurred even when the eyes were at rest. These tonic and clonic contractures are, as a rule, accompanied by diplopia. Some patients complain of diplopia of short duration, recurring several times in the day, attributable to slight contractures of this kind, and so slight and transitory that no trace of them can be found upon systematic examination.

The contractures which affect movements of convergence or the associated movements of direction possess the common characteristic that they affect both eyes simultaneously, this peculiarity being almost constant in ocular disorders of an hysterical nature. There are exceptions, examples of which are furnished by the ocular contractures included in unilateral blepharospasm. In cases of this kind the disturbance may be monolateral, but it shows itself readily on the other side when the patient attempts to fix with the eye affected by the blepharospasm, which eye is generally deviated upward and inward. Photophobia, being usually intense, makes a proper examination difficult. Moreover, the sensitiveness to light appears to be the immediate cause of the contracture, which may disappear when the patient is in darkness, thus explaining its localization in one eye by reflex action.

Ocular irritants, or those which affect any branch of the trigeminus, may develop or perpetuate hysterical contractures, the chief cause of which, however, is the predisposition of the patient. In several cases we have seen the contracture disappear after the removal of carious teeth, as in a case cited by Terrier and Mengin.

Apropos of monocular contractures, I must call attention to a possible source of error in diagnosis, a point that I indicated fifteen years ago. In certain cases of paralysis, and especially incomplete palsy of one muscle, the associated muscle of the other eye may become the seat of a spasm so pronounced that the patient complains of the eye which is healthy much more than of that which is the seat of the paralysis. As the paresis is sometimes very slight, it may easily be assumed that the symptoms arise from a contracture affecting one eye, although they are really provoked by a paralysis in the other. I cannot here discuss all the clinical varieties of these spasms of associated muscles, although I have elaborated them in a monograph.[1]

Hysteria is pre-eminently the disease of contractures, but when it is found in presence of a contracture of the kind above referred to, I think we may with certainty exclude its influence. I believe, in fact, that spasm

[1] Spasmes et paralysie des muscles de l'œil, Gazette hebdomadaire de médecine et de chirurgie, 1877.

of an associated muscle is peculiar to peripheral paralysis, and it is almost certain that peripheral paralyses do not exist in hysteria.

Paralysis of the third pair and contracture limited to one muscle or to the territory supplied by one nerve probably do not exist in hysteria. This is the conviction at which I have arrived after long-continued observation of hysterical patients, and since I began to appreciate the sources of error in diagnosis to which we are exposed. Of these causes of error the chief is the possible coexistence of an organic disease with hysteria. In dealing with cases of hysteria—a disease that is continually presenting new phases—it is always prudent to make certain reservations, as well as to take into account the observations of others. For example, Samelsohn, who is well known to be a careful observer, stated to me that he had seen a case of hysterical paralysis of the sixth pair.

I think, however, that an analysis of the facts along the lines I have laid down will enable us to assert that alleged cases of paralysis of nerve-pairs are probably not paretic in character; that is, they are more likely to be paralyses or contractures of associated movements, either of direction or of convergence, or of both together.

If we examine from this stand-point the case which was the occasion of Borel's monograph, and which, by the way, furnishes one of the few examples of a case-record satisfactorily reported, we must doubt the author's diagnosis. He gives the following example of "true hysterical paralysis of the external rectus." The characteristics of this case were the variability of the symptoms and the predominance of motor disturbance, at one time on the right, at another on the left side, which, as I have just said, are not rare in hysterical conjugate affections. In the field of fixation shown on page 535 of Borel's monograph I find that the lateral excursion was forty-three degrees inward and thirty degrees outward in the right eye and thirty-seven degrees inward and forty-one degrees outward in the left eye. This certainly does not indicate a weakness limited to the right rectus muscle, but points to failure of all the associated movements to the right.

The author, moreover, makes this formal statement: "There is, then, a paresis of the rectus externus of the right eye with weakness of the internal muscles." There could not, however, have been insufficient interni, since, in the lateral excursion to the left, the inward movement of the right eye is given as forty-three degrees, which is normal; that is to say, there is simultaneously with a defect of the associated movements of direction a defect of the associated movements of convergence, the symptoms of which he gives very explicitly in the report of the case. Indeed, the writer finds repeatedly emphasized the following statement: "At a distance, in the median plane, homonymous diplopia of fifteen degrees was observed; nearer, single vision was established; still nearer, crossed diplopia appeared." These are symptoms which the writer has given as characteristic of contracture of convergence.

From the cases of paralysis of the third pair attributed to hysteria must be eliminated those of which ptosis is a symptom. They are examples of what the writer has described as pseudo-paralytic ptosis, the true nature of which he will further discuss, but which cannot in every instance be considered as a partial paralysis of the third pair. There are but a few observations of this form of paresis, save the incomplete report of Guttmann[1] and that which the writer made in 1882 and which appears in the monograph of Richer. In Guttmann's case there were a sensitive and sensory left hemianæsthesia, motor paralyses, and convulsive attacks. The examination of the eyes, made by Leber, revealed a bilateral amblyopia and concentric contraction of the visual field without achromatopsia, more marked on the right side. On both sides there were paralysis of the oculo-motorius; complete right, partial left ptosis; and paralysis of the left internal rectus and of both superior recti. The pupils were normal.

The following is a summary of the case which the writer observed. A girl, seventeen years of age, was seen in the service of Charcot. After convulsive seizures, which occurred September 8, 1882, ptosis of the right lid, with diplopia and dilatation of the pupil, appeared. After treatment, she left the hospital much improved, though later there were additional convulsions and a return of the symptoms. She entered the Salpêtrière on October 14, when the writer found the following notes: "The pupil is dilated *ad maximum*, and there is paralysis of accommodation,"—a condition that made him suspect atropine mydriasis. Examination of the eye, however, revealed other symptoms of paralysis of the third pair. In addition to a slight ptosis, there was a crossed diplopia in the left half of the field of vision that presented the characteristics of an incomplete palsy of the internal and inferior rectus muscles. There was contraction of both visual fields, more pronounced on the right side. There was no fundus lesion. Right hemianæsthesia was present." This case, like Guttmann's, was undoubtedly one of paralysis of the third nerves occurring in an hysterical subject. It remains to be determined whether the paralyses were themselves hysterical. So far as the writer's patient is concerned, her age and the coincidence of the ocular disorder with the convulsive attacks argue in favor of hysteria. Still, the observation of this case, extending over eleven years, is not sufficient for conviction. The writer persists in the belief that a well-marked paralysis of the third pair in an hysterical subject should make us suspect other influences and the coexistence of two separate diseases. The following history of a patient seen by him goes to justify this opinion. A complete report of the case, with autopsy, was published by Blocq and Onanoff.[2] The writer first saw the patient in the service of Professor Debove, and afterwards in that of Charcot. The patient was affected with bilateral paralysis of the third

[1] Ein seltener Fall von Hysterie, Berliner klinische Wochenschrift, 1869.

[2] Sur un cas d'association tabéto-hystérique, suivie d'autopsie, Archives de médecine expérimentale, May, 1892.

nerve. He exhibited a double external strabismus. The paralysis was symmetrical, and had produced entire abolition of all the muscular movements controlled by the oculo-motorius. In addition, he had contraction of both visual fields without lesion of the fundi, marked hysterical disorders, and ocular paralyses apparently not referable to another disease. Nevertheless, in the presence of both Debove and Charcot, the writer expressed doubts as to the hysterical origin of these paralyses, and the autopsy demonstrated that he was right. There was found post mortem a pronounced degeneration of the third pair, due to tabes superior.

(*e*) *Blepharospasm.*—Blepharospasm and amaurosis are the earliest known hysterical ocular manifestations. The writer has stated that Hocken (1844) believed that blepharospasm always accompanies amaurosis. This error, opposed by Landouzy, shows that hysterical blepharospasm did not escape the notice of the early observers who made a scientific study of this neurosis.

We distinguish two kinds of blepharospasm, the tonic and the clonic form. In the tonic variety the eye is closed convulsively, as in contraction of the orbicularis due to strong voluntary effort. The spasm is sometimes limited to the orbicularis, but may extend to the muscles of the face and the neck. Tonic blepharospasm is usually monocular, but the other eye may be affected. If attempts are made to open the lids, we shall find that there is more or less intense photophobia, that the eyeball is convulsively displaced in different directions, and that these spasms, which are provoked by the impression of light, generally extend to both eyes.

We often discover spasm of accommodation along with the blepharospasm, but its determination is sometimes impossible. Slight degrees of blepharospasm may exist without amblyopia (which they sometimes precede), but more frequently amaurosis appears to accompany the condition. It has been alleged that a well-marked blepharospasm may mechanically produce amaurosis by pressure of the lids upon the globe. Such cases should be submitted to a critical examination to exclude possible hysterical element.

Clonic blepharospasm manifests itself by more or less frequent blinking of the eyelids, which ordinarily affects both eyes. This blinking is but one phase of the hysterical seizures, although it has been observed apart from such attacks. Still, clonic blepharospasm is not, strictly speaking, an hysterical stigma. It is of the nature of tic, and, like all the tics, is rather a mark of mental unsoundness. At all events, it is frequently observed in non-hysterical cases.

In hysteria the lid-movements may be deranged in other ways. In 1881 I brought to Charcot a child, twelve years of age (a report of whose case is given in Paul Richer's monograph), who was attacked by an apparently paralytic closure of the lids. There was also convergent strabismus, and neither the closure of the lids nor the strabismus was of paralytic origin. As for the drooping of the lid, I declared that it was

hysterical, and that it was merely a variety of what I term pseudo-paralytic ptosis. Charcot, who gave a clinic on the patient, recalled certain similar cases in which the diagnosis of paralytic ptosis made by distinguished ophthalmologists had not misled him.

Pseudo-paralytic ptosis resembles levator paralysis in this particular, that the upper lid droops without wrinkling of the palpebral skin, and when the patient is ordered to open the eye the frontalis muscle generally contracts as in paralysis. Still, if the lid be raised with the finger, it falls more quickly than in paralytic ptosis. There is, besides, almost always a certain degree of photophobia. Finally, the lid, which the patient cannot move voluntarily, is raised at certain times when the attention is forcibly diverted by some unforeseen circumstance, or in the evening, when the surrounding light is less intense. Charcot has called attention to the important point that in unilateral hysterical ptosis the eyebrow is lower than on the healthy side, as opposed to that which obtains in true paralytic ptosis, in which the eyebrow is more elevated. The diagnosis is also facilitated by the amblyopia that habitually accompanies the hysterical variety.

The lids may also show a more singular disorder. In addition to the impossibility of opening the eye and of raising the upper lid, inability to close the lids completely, as in orbicularis paralysis, may be observed. Case No. 67 in Morax's report illustrates this. The patient seemed to be attacked simultaneously by paralysis of the levator palpebræ and of the orbicularis. I say he "seemed to be" so affected, because we must not lose sight of the fact that he had no muscular disease, but exhibited rather an affection of the nerve-centres that preside over two antagonistic movements. Pseudo-paralytic ptosis may be replaced by marked blepharospasm or may follow it, as in a case reported by Zehender.[1] This is an additional proof that it is not a true paralysis. The writer cannot avoid calling attention to a certain analogy between this anomaly of the lid-movements and spasm of accommodation which we have considered. In both cases the nervous disorder may affect two antagonistic actions, and at one time there may be the characteristics of paralysis, at another those of contracture.

Blepharospasm may show itself as the result of diverse influences, and may follow a seizure or an emotional disturbance, like all other hysterical contractures. It is often developed by irritation of the eye, integument, or mucous membranes. Lasègne[2] has cited an instance of a young girl who got some grains of sand into the eye, and had contracture of the lids that persisted for several months after all irritation of the conjunctiva had disappeared. In Zehender's case, to which reference has been made, the blepharospasm could be brought on or exaggerated by pressure over certain parts of the body,—in the neighborhood of the first cervical vertebra or in the left sternal region. In a case reported by Seeligmüller,[3] spasm of the

[1] Klinische Monatsblätter für Augenheilkunde, 1875.

[2] Des hystérics périphériques, Archives générales de médecine, June, 1878.

[3] Klinische Monatsblätter für Augenheilkunde, 1871.

orbicularis could be produced at will by pressure of the inferior maxilla upon the posterior superior molars. In other patients, on the contrary, pressure upon different points stops the blepharospasm. These facts have been well known to ophthalmologists since Graefe's report.[1]

In tonic blepharospasm the muscles of the eyeball always seem to be involved in the contracture. After the spasm has persisted for a long time, and is finally cured, so that the patient can open his eyes freely, he may complain of diplopia. I have seen a case in which the patient had a homonymous diplopia exaggerated on looking to the left, and in which I certainly should have made the diagnosis of paresis of the left sixth nerve, had I not known from the patient's previous history that he suffered from spasm, which, as I have said before, may be limited to one eye or to one muscle when it accompanies blepharospasm.

Among the concomitant symptoms of blepharospasm must be noticed the anæsthesia of the palpebral skin pointed out by Gilles de la Tourette, the epiphora, and the periocular pains, which may become spontaneous or be induced by the slightest touch.

## SEMEIOLOGICAL VALUE OF THE OCULAR DISTURBANCES OF HYSTERIA.

The writer is more and more convinced that the ocular disturbances just described are peculiar to hysteria. Several of them, even considered separately, appear to be peculiar to this neurosis. Especial mention will be made of these: the visual fields for red and for other colors are more extended than the field for white; dyschromatopsia is characterized by a peculiar inversion of the color-field; and there is spasm of accommodation and of convergence,—a temporary condition in children. Primary contractures of the associated movements of direction, as well as the tonic form of blepharospasm, even when they are of reflex origin, are probably always hysterical. Certain cases of conjugate deviation in cerebral apoplexy and in acute affections of the brain must, of course, be excepted. Pseudo-paralytic ptosis is always due to hysteria. Dissociation of the associated movements in convergence and of direction is not peculiar to hysteria; neither is dissociation of the movements determined by the different factors in convergence, because we observe that symptom in the early stages of certain forms of strabismus; but it is, nevertheless, of considerable value in the diagnosis of hysterical disorders. Dissociation of voluntary and reflex movements in ophthalmoplegia, and defects of the associated movements of direction, such as are seen in certain forms of muscular tremor (astasia-abasia), are probably characteristic of hysteria.

Surprise may perhaps be expressed at not including concentric limitation of the visual field in this list, that symptom having been almost exclusively insisted upon as characteristic of retinal anæsthesia or hysterical amblyopia. Concentric narrowing of the visual field without lesion of the ocular

[1] Klinische Mittheilungen ueber Blepharospasm, Archiv für Ophthalmologie, 1871.

fundus is certainly a symptom of great practical value in the diagnosis of hysteria, but it is not peculiar to that neurosis. It is found in some cases of cerebral tumor without neuritis, or it may result from intra-cranial pressure associated with certain forms of extensive cerebral meningitis. According to Charcot, it is found in lesions of the posterior portion of the internal capsule. Finally, it is not infrequently observed in epilepsy.

It is especially in epilepsy that the differential diagnosis is difficult, not only as regards the ocular disturbances, but in respect of other symptoms. A radical distinction must first be established between those temporary ocular symptoms that follow epileptic attacks and other disorders of a more permanent character. It is well established that narrowing of the visual field may follow epileptic seizures and persist for a considerable time thereafter. In eighteen subjects observed after such attacks, Féré found a contraction of the visual field, and this led him to believe that all epileptic manifestations associated with loss of consciousness are invariably followed by amblyopia, which may be discovered if one examines the patient immediately. It is also certain that this temporary amblyopia, which may disappear rapidly when there is but one attack, may persist much longer when the attacks are repeated. In some patients at the Salpêtrière the writer has seen it persist for nearly twenty-four hours after an attack.

The amblyopia may amount almost to amaurosis, and may last for a longer or shorter time after the attack. The writer may add that we may have temporary amauroses, unaccompanied by convulsive attacks, which are undoubtedly manifestations of epilepsy. He communicated a case of this kind to Antonelli,[1] who utilized it in his article on "Transitory Amblyopia," while Gayet also reported to the Congress of the French Society of Ophthalmology, in 1893, a case which he considered to be of an epileptic nature.

According to Féré, the amblyopia which follows an epileptic seizure is characterized by a reduction of vision in all its forms,—the visual acuity as well as perception of white and of colors. The writer made similar observations of the case reported by Antonelli, and was studying the symptoms at the moment the amaurosis disappeared. Until the contrary is proved, it may be stated that the amblyopia which follows the attacks is not accompanied by dyschromatopsia, especially the peculiar inversion of the color-field found in hysteria. The amplitude of accommodation may be contracted after the attacks, but the writer has not noticed the typical spasm that is so characteristic of hysteria.

It is evident that epileptic seizures, and probably every cerebral disorder that is accompanied by loss of consciousness, may occasion a temporary amblyopia characterized by enfeeblement of all forms of the sight-sense, the dominant symptom of which is concentric narrowing of the field of vision. It is also certain that this amblyopia may persist for a variable

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[1] Archives de neurologie, 1892.

time in the interval between attacks, especially when the latter are repeated. These sensory anomalies following such attacks are not peculiar to vision. Smoler, Gowers, Russell, and Bennett have described disturbances of the sensibility of the skin and other organs.

The really important question is whether there is a permanent limitation of the visual field in epilepsy as there is in hysteria. I shall not consider cases in which only contraction of the color-field has been noted, as in the histories reported by Pichon. Nothing is more difficult than to make a diagnosis from narrowing of the color-field alone, especially when the inversion of which I have already spoken is not present, and when there is no limitation for white.

Oppenheim and Thomsen [1] found contraction of the visual field in one-third of ninety-four epileptics. Such a result, as may be imagined, very much lessens the value of a perimetric examination in the differential diagnosis between hysteria and epilepsy. To verify this, the writer requested one of his pupils, M. Hitier,[2] to examine the epileptics in the hospital service of Charcot. Of eighty-seven cases, he found permanent contraction of the visual field, "not due to any appreciable cause but epilepsy," in only three. We know that epilepsy and hysteria may coexist in the same patient, as Charcot has shown. We ought, of course, to know how to make the distinction in these cases. This may be said: the writer can affirm, after numerous examinations both at the Salpêtrière and in his private practice, that, although contraction of the field is present in certain cases of epilepsy, the condition is still of considerable value in the differential diagnosis of these two neuroses. The existence of narrowing of the visual field in epileptics makes it all the more necessary to base an opinion not upon that sign alone, but upon the presence or absence of all the symptoms which have just been described as characterizing hysteria,—a picture which epilepsy would certainly not present.

These symptoms—the amblyopia as well as the motor disorders—are observed in certain cases of nervous excitement developed after injuries, and especially after injuries accompanied by violent mental disturbance or nervous shock. Charcot has demonstrated the identity of these symptoms with those of hysteria, and has described them under the name of *traumatic hysteria.* This identity was immediately conceded in America by Walton,[3] who had studied nervous diseases in the service of Charcot at La Salpêtrière, and by Putnam.[4] In Germany, on the other hand, Oppenheim and Thomsen [5] have attempted to make these nervous symp-

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[1] Archiv für Psychiatrie und Nervenkrankheiten, 1884.

[2] De l'amblyopie liée à l'hémianesthésie et spécialement de l'amblyopie hystérique, Thèse de Paris, 1886.

[3] Hysterical Anæsthesia brought on by a Fall, Archives of Medicine, 1882, vol. x; Boston Medical and Surgical Journal, 1884.

[4] The Medico-Legal Significance of Hemianæsthesia after Concussion Accidents, American Journal of Neurology and Psychology, 1884.

[5] Arch. de Westphal., vol. xv.

toms a special disease, to which they have given the name *traumatic neurosis.* Charcot's belief is, the writer thinks, now accepted by every one, even by the later German school represented by Bruns, Möbius, Freund, etc. In the recent monograph of Frankl-Hochwart and Topolanski, already cited, a work emanating from Nothnagel's clinic, similar opinions are held.

Those who may still have doubts about the matter are referred to the thesis of Berbes,[1] to that of Guinon,[2] to the treatise of Gilles de la Tourette, already cited, and finally to a recent and very complete review of the question by Bruns.[3]

If doubts still remain as to the identity of hysteria with the traumatic neurosis, the ocular anomalies will certainly dispel them. These anomalies are identical in both cases; and, as they are generally much more easily recognized and demonstrated than those of other nervous disorders, the writer has many times had occasion to prove their importance from a medico-legal stand-point. In this connection he again insists upon the necessity not only of measuring the visual fields and determining the central acuity, but also of establishing the presence of those accommodative and convergence anomalies which we know may exist without amblyopia.

Traumatic hysteria suggests a study of the so called reflex amblyopia and of those ocular symptoms that are developed as the result of some peripheral irritant. This may have its seat in the circumference of the orbit, in some twig of the trigeminus, or even at a more distant point,—in the intestinal canal, for example. The writer has reported cases of amblyopia, occurring after traumatism of the orbital margin, which presented all the characteristics of hysterical amblyopia, and has noted an instance in which a visual defect of the same nature disappeared after the evacuation of a tape-worm.[4]

I have already stated that ocular contractures, blepharospasm in particular, which seem to be dependent upon peripheral excitation are probably always hysterical, the peripheral irritation acting as a hysterogenic zone. Such cases are coming to be considered more and more as examples of hysteria. This is a question which cannot be discussed at length here, but I wish to call attention to one form of hystero-traumatic amblyopia simulating sympathetic disease that might lead the ophthalmologist into error. In 1884 I observed, in Charcot's service, a patient who had been treated for a long time for disease of the right eye. The left eye, the sight of which had been destroyed by an accident, had been enucleated. The excision of the injured eye had not affected the vision of the right eye, in which all the characteristics of hysterical amblyopia were found. The patient eventually presented the entire grouping of symptoms of hysteria (*la grande hystérie*).

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[1] Hystérie et traumatisme, Paris, 1887.

[2] Les agents provocateurs de l'hystérie, Paris, 1889.

[3] Neuere Arbeiten ueber die traumatischen Neurosen, Schmidt's Jahrbücher, 1893.

[4] Amblyopie hystéro-traumatique, Société d'ophthalmologie de Paris, June, 1889.

A second patient, who had the left eye enucleated in 1887, on account of a hunting accident, consulted the writer in April, 1893, for an intense neuralgia extending over the entire right side of the face, together with disturbance of vision in the remaining eye. It was ascertained that in this eye there was concentric narrowing of the visual field, with practically normal acuity and no fundus-changes. In addition, there were spasm of accommodation and monocular diplopia. The patient was reassured with regard to any immediate danger of sympathetic disease, a result which some of the writer's colleagues had feared. Six months later there was no trace of sympathetic symptoms, the limitation of the field remaining unchanged.

The third case was that of a railway employee, forty-nine years of age, who was brought to the writer's clinic in April, 1893, on account of a destructive injury to the left eye. The injured eye was painful, and the patient complained of trouble in the right eye, in which there were no visible changes. The left eye was enucleated one month after the accident, but the symptoms in the right eye did not disappear. Before he obtained an indemnity from the railway company, he was suspected of malingering. However, the writer examined him carefully, and discovered a typical hysterical amblyopia, characterized by concentric contraction of the visual field to thirty degrees, with the field for red more extended than that for white; vision equalled 5/25. There was very little spasm of accommodation, but this is rarely found at the patient's age. As hysterical signs that cannot be simulated, the writer again calls attention to anæsthesia of the pharynx and epiglottis, which in this case could be touched by the finger without provoking reflex symptoms. The visual defect in the right eye was developed soon after the accident, and it is noteworthy that the patient dreamed every night that he was going to lose his sight.

Here, then, are three cases of unilateral hysterical amblyopia following injury to the other eye, observed in men in whom one would not be likely to suspect hysteria; and although the symptoms simulated sympathetic disease, they had, in reality, no connection whatever with ophthalmia migratoria. Moreover, the conditions were not at all modified by enucleation.

A case reported by Kalt[1] is of interest. A woman, twenty-three years of age, who had lost the left eye, noticed a serious diminution of vision in the right eye: V. = 1/20. There were no fundus-lesions. Enucleation of the left eye caused a great improvement of vision, which a month later equalled 2/3. Kalt considers the visual disorder hysterical, but the writer must say that the amblyopia presented none of the characteristics of hysteria. The narrowing of the visual field, in particular, was wanting.

Hysteria may be observed in the same subject simultaneously with

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[1] Amblyopie hystérique sympathique, Bulletin de la Société d'ophtalmologie de Paris, 1892.

another neurosis or with an organic disease. The conditions most frequently associated with hysteria are epilepsy, Basedow's disease, disseminated sclerosis, Friedreich's disease, tabes, syringomyelia, poisoning from lead, alcohol, etc., and syphilis. Very often the two combined diseases can be recognized from their ocular manifestations, an experience which the writer has often had at the Salpêtrière, and in which he does not remember that additional medical examinations showed that he had made an error in diagnosis.

Of seventy-nine cases of hysterical affections of the eye which figure in the report of the writer's clinic for 1888, made by his assistant, M. Morax,[1] in nine instances the condition was associated with some other disease. The ocular symptoms accompanying each disease are indicated in the report. The diseases with which hysteria was associated in the nine cases were as follows: disseminated sclerosis, one case; tabes, two cases; Friedreich's disease, three cases; syphilis, one case; Basedow's disease, one case; and alcoholism, one case.

The writer's present assistant at the Salpêtrière, Dr. Koenig,[2] made a report upon the subject to the 1893 meeting of the French Ophthalmological Society.

Buzzard, who with Charcot pointed out the frequent coincidence of disseminated sclerosis with hysteria, has also insisted upon the possibility of distinguishing between the visual disorders proper to each disease.

The writer regrets that he cannot discuss here the differential diagnosis between the ocular disturbances of hysteria and those of the disease with which it may be associated. The distinction, he repeats, must be reserved for the ophthalmologist who is familiar with that kind of work. The coexistence of hysteria with another disease is likely to give rise to the error of attributing, for example, contraction of the visual field or some other hysterical disorder to an organic lesion. Thus it was that Dejerine and Tuilant regarded concentric limitation of the visual field as a symptom of syringomyelia, when in reality it was caused by concomitant hysteria. In about ten patients with this disease whom the writer examined he found contraction of the field once, this patient, however, failing to present other signs of hysteria. In nine cases recently examined by Hoffmann[3] the narrowing of the visual field was not once observed.

When concentric limitation of the field, or any other symptom which the writer has considered, is observed in patients suffering from organic disease, we should not hastily attribute it to the latter affection, but should always bear in mind the possible coexistence of hysteria with organic lesions.

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[1] Archives de neurologie, 1889.

[2] Troubles oculaires dans les associations hystéro-organiques, Bulletin de la Société française d'ophtalmologie, 1893, p. 480.

[3] Deutsche Zeitschrift für Nervenheilkunde, iii. S. 1, 1883.

## TREATMENT.

As the ocular disorders in hysteria are only symptoms of a general disease, the treatment is, above all, that of the neurosis itself, and should be chiefly based upon a good mental and physical hygiene. The practice of suggestion, whether it be carried out while the patient is under the influence of hypnotic sleep or whether he be awake, may be of great service. This treatment, however, belongs especially to the neurologist; and as the writer has not had sufficient experience with it, and as it is not in his department, he will refrain from speaking further of it. Outside of general treatment, the ophthalmologist may intervene very effectually in many cases to palliate, if he cannot remove, the ocular symptoms of hysteria.

We are consulted especially on account of asthenopia, photophobia, ocular pain, and blepharospasm. As the treatment of these several affections has been considered elsewhere in this work, and as the writer must abridge his article, he will confine himself to a few general indications.

Hysterical asthenopia is most frequently due to anomalies of accommodation. Weak convex glasses relieve many patients, not only when the disorder appears in the form of an accommodative insufficiency, but also when there is a high degree of spasmodic myopia. Glasses of 1 or 1.5 D. strength are most often indicated for near-work, but one cannot mathematically deduce the formulæ of the lenses from the state of the refraction. Weak glasses must first be prescribed, and their strength, as occasion demands, gradually increased.

For extreme degrees of spasm of accommodation atropine is useful. This spasm, which readily disappears under medication, reappears, it is true, when the mydriatic is discontinued, but it is usually less marked than before, especially if other means be resorted to for preventing its recurrence, such as convex lenses, prisms, or smoked glasses.

When insufficiency of convergence is the chief symptom, prisms of two or three degrees, base in, are useful. As the anomaly of convergence in hysterical patients is almost always accompanied by accommodative weakness, a combination of convex glasses with prisms may be found necessary, and often renders important service. There can be no doubt, moreover, that by the use of prisms, base in, a certain amount of relief is given from the accommodative spasm. Conversely, convex glasses may act upon the convergence. Here, again, we cannot deduce the strength of the prisms from the state of the amplitude of convergence, weak prisms, for example, sometimes relieving patients in whom it is greatly reduced.

Prisms alone, worn constantly, are indicated when the anomaly of convergence is accompanied by vertigo. In some patients the writer has observed vertigo, almost amounting to agoraphobia, disappear almost immediately upon the employment of prisms.

When photophobia is the dominant symptom, recourse should be had to smoked or tinted glasses of various colors. As a rule, however, patients

make use of such protective glasses before consulting a physician. The writer insists not only that smoked glasses are useful on account of the relief they give the patient, but also that they diminish the amount of accommodative spasm as well as the contraction of the visual field. In other words, they exert a direct action upon the amblyopia.

As to those ocular and periocular pains which are not due directly to an affection of accommodation or convergence, treatment is uncertain; sometimes it fails, sometimes it is successful. It may fail because the pains, being essentially psychic in character, are not amenable to treatment like other neuralgias. The successes are probably due to suggestion, to which certain patients are particularly susceptible.

It cannot, indeed, be doubted that in the treatment of ocular hysterical symptoms suggestion has much to do with a good result, even when the physician does not suspect it. This is true even of cases of blepharospasm cured by surgical interference. Probably section and stretching of the supra-orbital nerve, recommended by von Graefe and Panas, as well as extirpation of a painful cicatrix, practised with success by Pflüger, produce their effect by suggestion alone. Some skin areas, and certain twigs of the trigeminus, may constitute true hysterogenic zones, and may act as starting-points for reflex actions whose pathological *rôle* may be as important as, if not more important than, that of the neurosis itself. On the other hand, blepharospasm is really an affection of central origin, and we ought not to resort to surgical measures until all other means have been employed,—namely, general treatment; direct or indirect suggestion; the continuous electric current, recommended by Pflüger, Seeligmüller, and Harlan; the magnets used by Harlan; instillations of cocaine, which have proved successful in Meyer's hands; massage as employed by Abadie; and the revolving mirrors of Luys, as recommended by de Wecker.

# EYE-AFFECTIONS DUE TO GRAVES'S DISEASE AND HERPES ZOSTER.

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## THE OCULAR SYMPTOMS IN GRAVES'S DISEASE.

ONE of the most constant and characteristic symptoms of Graves's disease, as one would infer from its alternative name of exophthalmic goitre, is an abnormal prominence of the eyes. Indeed, the patient's attention is often first directed to her "staring eyes," though, as a rule, the exophthalmos is preceded by irregular heart-action, digestive trouble, or the sense of fulness in the neck which the enlarging and pulsating thyroid gives.

The staring appearance of the eye is due to one or both of two causes: (1) actual projection forward or proptosis of the globe, and (2) abnormal increase in vertical measurement of the palpebral fissure, so that more of the sclerotic is exposed than is normally the case. The pushing forward of the eyes may be of rapid onset, and the more acute it is the greater is the risk of defects of nutrition—ulcers, or even sloughing—of the cornea. The exact reason for the proptosis is still undecided. Most writers invoke the arterial dilatation in the orbit, leading to an overgrowth of the fatty and connective tissue, as a sufficient cause, though another suggested one is a state of spasm of the unstriped muscle found in the orbit (especially that crossing the spheno-maxillary fissure), as well as in the eyelids themselves.

Persistent slight retraction of the upper lids (known as Stellwag's sign) is present in about eight out of every ten cases, and impairment of consensual downward movement of the upper lid with that of the globe (von Graefe's sign) is also seen in a large proportion. Slight depression or retraction of the lower lids has been noticed in a very few cases. Koller and Jessop have shown that similar phenomena on a small scale may be produced by applying cocaine to the conjunctiva, and it seems most likely that this is due to a direct stimulation of the sympathetic nerve-endings, and a consequent contraction of the orbital muscles supplied by these nerves.

Von Graefe's sign, already alluded to, is investigated in the following manner. The observer faces the patient, and directs her attention to an object which is made to travel slowly down in front of the face. Under normal conditions the upper lid follows in the most even manner the globe

as it is turned downward; but in most cases of exophthalmic goitre the lid will be seen to halt, leaving a widening gap between its lower border and the upper corneal margin. Spasm of the involuntary muscular fibres in connection with the levator palpebræ is certainly the most reasonable explanation for this interesting phenomenon, which, it must be noted, is not present in all cases of Graves's disease, and has occasionally been met with when all other signs of that disease were absent. (Hughlings Jackson.)

With the protrusion several other symptoms may be observed. The patient closes the lids with difficulty, and in bad cases they remain open during sleep: hence the production of corneal nebulæ, or ulcers, is readily understood, but is, fortunately, rare. Sometimes, however, complete opacity or sloughing of the cornea has led to blindness. In the latter cases the destructive process resists all local treatment, unless it yields to tarsorrhaphy or stitching the lids carefully together over the globes. It would seem that the more rapidly the protrusion develops the greater is the risk to the cornea.

With regard to rarer symptoms, we have to note that diplopia is sometimes experienced, and that even marked paralysis of one or more of the muscles moving the globe has been observed. Thus Bristowe records one case in which complete one-sided ophthalmoplegia externa supervened upon the protrusion.

One of the most distressing symptoms consists in attacks of severe pain in the eyes, accompanied by profuse scalding lacrymation. Occasionally the eyeball may be suddenly dislocated forward, and this may recur repeatedly. Loss of the eyebrow and eyelid-hairs has been noticed by Burney Yeo. On auscultation of the orbit a more or less continuous bruit is sometimes audible.

With regard to the condition of the pupils in Graves's disease there is not much to be said. Moderate dilatation has been noticed in some cases; but the pupils always respond well to direct illumination and contract on convergence of the eyes. These statements hold true for the great majority of cases, though, of course, the occurrence of perforating ulcers may lead to anterior synechiæ and thus limit the pupillary action. Further, iritis is by no means an infrequent result in severe cases.

Ophthalmoscopic examination, even in the worst cases, reveals little or nothing abnormal. It might be expected that the retinal vessels would share in the general dilatation of the orbital ones; but, although venous fulness and pulsation may be present, arterial pulsation is practically never observed in the retina in cases of Graves's disease. Optic atrophy has very occasionally resulted, apparently due to the stretching of the optic nerve.

Graves's disease, as is well known, is far more frequent in women than in men, although the latter sex is by no means exempt. It is a curious fact that von Graefe observed a great preponderance of men in the cases of destructive inflammation of the cornea following exophthalmos which he collected. This has never been confirmed by other observers. Out of twenty-

five similar cases tabulated by Jessop (including several of von Graefe's) only six occurred in men.

As regards the age of the patients, the period between twenty and forty years of age is the most liable ; but severe exophthalmos has been known to occur as early as four years and as late as fifty-six.

Unilateral exophthalmos has been observed in several cases associated with pulsating enlargement either of the whole thyroid gland, or of that half situated on the same side, or even of the opposite half. Thus, in one case recorded by Burney Yeo,[1] enlargement of the right half of the thyroid coincided with prominence of the left eye, and when right exophthalmos came on, the left lobe of the thyroid enlarged.

With regard to the *diagnosis*, it must be remembered that whilst the advanced cases can hardly be mistaken for any other disease, others may be met with in which the symptoms are but slightly marked, and that a mistake may in these latter readily occur. In other words, there are all degrees of Graves's disease, ranging upward from those in which there is only a slight undue exposure of the sclerotics and forward projection of the globes, with palpitation of the heart and fulness of the vessels of the neck. In these slight cases, which may or may not go on to the higher grades, the symptoms vary from time to time, and may be practically absent so long as the patient is not under the influence of excitement or strong mental emotion.

It is of importance to recognize these early cases of Graves's disease, which may come first under the ophthalmic surgeon's care, since treatment is then most likely to be successful.

**Prognosis.**—With regard to the prognosis of the disease, of which the eye-symptoms form but a part, one fact has been clearly established,—that cases of even great severity may slowly but completely recover, and all traces of the exophthalmos ultimately disappear. This fortunate result may be independent of any drug treatment, and in some cases has followed a complete change of climate and habits of life. Occasionally the occurrence of pregnancy has had a favorable effect upon the disease, but this is by no means constant.

With regard to the eye-symptoms, we may say that the more rapid the development of the globe-protrusion the greater is the risk of corneal trouble. It is just in these cases of rapid onset that complete opacity or sloughing of the cornea may develop, leading to complete blindness.

Once severe inflammation of the cornea has supervened, local treatment is, as a rule, unsuccessful.

**Treatment.**—Since it is generally admitted that the chief danger to the eye in Graves's disease results from inability to close the lids, and the consequent loss of their protective power, the most hopeful measure of local treatment would appear to be the operation of tarsorrhaphy, or suturing the lids together.

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[1] British Medical Journal, 1887, p. 820.

This should, however, be reserved for the worst cases, and, if it becomes necessary, it should be done thoroughly by removing a considerable part of each lid-margin. It should be noted that some consider the operation inadvisable if the patient is past middle age, and that there appears to be a special risk in administering anæsthetics to the subjects of Graves's disease. In cases that have recovered with leucomata iridectomy may subsequently be required. In treating the mild cases of corneal ulceration frequent bathing of the eye with a warm antiseptic solution is usually of benefit.

A few words must be said as to the general treatment of Graves's disease.

Change of climate (note that high altitudes are said to be injurious), avoidance of all excitement so far as is possible, and attention to general health are perhaps the most important measures.

Of the host of remedies advised in the treatment, belladonna taken internally has been most generally found useful. Phosphate of soda in large doses is advocated on theoretical grounds, and several cases of improvement under its use have been reported. If the case is attended with much mental excitement or distress, sulphonal in from ten- to twenty-grain doses may be found useful. Aconite, quinine, digitalis, and iron have all been extensively tried, with occasional success.

Several observers have reported favorable results from the administration of thymus gland (preferably obtained from the calf, and administered in doses of from half an ounce to an ounce three or four times a week). The improvement was shown not only in the ocular symptoms but also in the heart's action, and the general condition. The treatment is based upon a supposed antagonism between the thymus and thyroid glands,—a condition which is by no means proved to exist; but since thymus gland feeding is practically harmless it is worthy of further trial in Graves's disease. The application of Leiter's ice-coil, or, better, of an "ice-collar," to the neck over the region of the thyroid gland has been known to cause marked diminution of the proptosis as well as of the vascular excitement.

Of late years many cases have been treated by partial excision of the thyroid gland or ligature of the thyroid arteries. Although it is perhaps premature to decide upon the value of these operations, it may be said that they are by no means devoid of risk in severe cases of Graves's disease, and that, on the whole, their results have been discouraging; nor can any valid reasons be given for expecting much improvement from them.

## HERPES OPHTHALMICUS.

For the last thirty years it has been known that when herpes occurred in the distribution of the fifth cranial nerve the globe on that side was liable to become involved.

It was pointed out by Mr. Hutchinson, and has since been confirmed by several observers, that if the skin of the nose be extensively affected by herpes there is a special risk of the iris and cornea becoming also inflamed.

This is to be explained by the fact that the sensory nerve supply of the iris (and some other internal parts within the globe) is derived from the long and short ciliary branches of the nasal trunk, which also reach the cornea. The sensory nerves of the eyelids, including those of the conjunctiva, caruncle, and lacrymal sac, are derived from several different branches of the fifth. Thus the upper lid on both surfaces receives twigs from the supra-trochlear and supra-orbital divisions of the frontal nerve as well as from the lacrymal; the lower lid from the infra-trochlear (branch of the nasal) and infra-orbital nerves. Hence, in herpes affecting one or other division of the ophthalmic or the superior maxillary nerve, we should expect the eyelids and conjunctiva to be more often inflamed than the iris and cornea. This is found to be the case. But it must be noted that we cannot lay down the rule that with herpes in the distribution of the nasal nerve on the face the iris or cornea is always involved. In several cases where herpetic vesicles have covered the side of the nose the eye itself has wholly escaped.

One remarkable fact about herpes occurring in the distribution of a sensory nerve is that the inflammation is of sudden onset and shows no tendency to spread; hence if the eye is affected it will be so about the same time as the development of the vesicles on the skin around. It may, however, happen that with severe herpes frontalis where there is much scabbing or an eczematous condition of the eyelids the conjunctiva becomes inflamed some days or a week or two after the onset of the skin-trouble. The same holds true in some cases of iritis with herpes.

As regards the pathology, there is every reason to believe that a neuritis, probably central, occurs. In more than one case of herpes zoster it has been demonstrated (by Charcot, Hebra, von Bärensprung, etc.) that the ganglion on the posterior root of the spinal nerve is inflamed or has hemorrhages into it.

From analogy one would expect in herpes frontalis that the Gasserian ganglion would be involved, and this fact seems actually to have been proved. (De Wecker.)

As to the cause of this neuritis, whether central or peripheral, we are still ignorant. It is suggested that exposure to cold may be one cause, and in favor of this view it is to be noted that the branches of the fifth nerve are peculiarly exposed, that they mostly pass through bony foramina, and that they are the favorite sites of neuralgia.

It is also certain that toxic influences may produce herpes: of these one of the best proved is arsenic; the internal administration of this drug has been known to produce herpes frontalis, though not nearly so often as herpes of the trunk, etc. Similarly, herpes frontalis has followed an attempt at suicide by charcoal fumes, and residence in a vitiated atmosphere has also been invoked as a cause.

In most cases, however, no explanation of this kind is forthcoming. We know that herpes of the fifth nerve occurs in both sexes with almost equal frequency, and that it attacks adults as a rule, though children are

not exempt. Many attempts have been made to prove that it occurs more frequently at one period of the year than another, but with only conflicting results. It very rarely relapses, but has been known to occur three times in the same individual. As regards its comparative frequency, herpes of the face occurred in sixteen cases out of a total of one hundred and sixty in which various parts of the body were affected, and intercostal herpes is more than fourteen times as frequent as the facial variety. (Greenough.)

The onset of herpes is usually preceded by pain, of either a darting or a boring character, in the area to be affected,—*i.e.*, the forehead, eye, etc. Sometimes this prodromal pain has lasted ten days before the first vesicles have shown themselves, though usually the period is very much shorter. A fact of more importance is that the neuralgia may last long after the eruption has developed, and perhaps when nothing remains but scars or pigmented patches. It would appear that the older the patient the more is this neuralgia to be dreaded and the longer does it persist. It is not only that the parts affected remain painful, but their sensation may be for long impaired; thus the cornea may be left anæsthetic, and therefore especially liable to ulceration from foreign bodies, etc.

As to the form of ophthalmic lesion accompanying herpes, one or more of the following symptoms may be present: first, swelling and perhaps vesication of the conjunctiva; second, vesicles of the cornea itself, ulcers, or even sloughing;[1] third, keratitis punctata, or iritis not truly serous (hypopyon is very rare). There may be tenderness over the lacrymal gland, with lacrymation. As a sequel of the corneal involvement nebulæ or troublesome phlyctenules may be left.

The eyelids during the active stage are nearly always congested and œdematous. Thus in the severe cases iritis or destruction of the cornea will occur. In mild ones there are merely ophthalmic irritability and photophobia, with slight congestion.

It has already been noted that herpes ophthalmicus is especially liable to complicate neuritis of the nasal branch; but it is seen with herpes frontalis and even herpes infra-orbitalis, though with the latter it is very rare.

Is herpes ophthalmicus ever complicated with lesions of the oculo-motor nerves? This has been much debated, and the answer seems to be that it is very rarely so complicated.

It is a fact of great interest that paralysis of the facial nerve (on the same side) may follow or accompany herpes facialis in the distribution of the fifth nerve. Rare though this complication is, several carefully recorded cases prove its occurrence beyond question. Of these we would especially refer to those reported by Letulle,[2] Verneuil,[3] and Tryde.[4] Strübing,[5]

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[1] This is to be expected only from the fact that gangrene of the skin sometimes occurs in herpes.

[2] Letulle, Archives de Physiol. norm. et pathol., 2d ser., t. ix. p. 162, 1882.

[3] Verneuil, Gazette de Paris, 1873, No. 20, *et seq.*

[4] Tryde, Schmidt's Jahrbücher, Bd. cliv. S. 273.

[5] Strübing, Deutsches Archiv für klinische Medicin, October, 1865.

Greenough, and others have recorded instances where the herpes had involved spinal nerves (the cervical set) and not the fifth itself. It is to be noted (1) that the motor paralysis has usually developed or been noticed as the skin-eruption was disappearing, or even after it had entirely healed; (2) that in herpes ophthalmicus, as in herpes of other regions of the body, there is often evidence of neuritis, disturbances of sensation (sometimes persistent anæsthesia), as well as pain; (3) that the motor paralysis is very slow to disappear, persisting, perhaps, when all traces of the cutaneous herpes have vanished except a few scars.

That herpes ophthalmicus is due to a neuritis of the first division of the fifth nerve there can be little doubt. But it is a form of neuritis which has little or no tendency to spread, differing in this respect most markedly from some other varieties of peripheral neuritis. The close proximity of the nuclei of origin of the fifth and seventh nerves in the floor of the fourth ventricle suggests a method of extension of inflammation which would account for these curious and rare cases of herpes ophthalmicus and facial paralysis. But do we ever see herpes of one branch of the fifth involving paralysis of the motor part of that nerve, or spreading from the ophthalmic division to the superior or inferior maxillary branches, once the eruption has definitely appeared? It would further appear that herpes ophthalmicus is very rarely indeed accompanied or followed by paralysis of any of the true ocular muscles.

It is true that in a case of Verneuil's,[1] where unilateral facial herpes was followed by incomplete facial paralysis (neither involving the forehead), there were at the same time strabismus and ptosis. But the latter two symptoms *were on the other side of* the face, and were doubtless due to injury of the third nerve by fracture of the cranial base, the injury having preceded all the nerve symptoms. In two cases, however, ptosis persisted after herpes ophthalmicus on the same side.[2]

**Diagnosis.**—It is a common thing for mistakes to arise in the diagnosis of herpes ophthalmicus, and this not merely at the onset of the attack. There is one disease with which it is frequently confounded, for both by the laity and by many medical men it is apt to be called erysipelas.

In its sudden onset, the pain which accompanies it, the vesication and swelling of the skin which are present at its height, and perhaps even in the constitutional disturbance which may accompany it, there are points of resemblance to erysipelas. But the points of distinction between the two are much more marked. Herpes ophthalmicus is strictly limited to the distribution of certain sensory nerves, occurs on one side only of the face, never spreads as does erysipelas, the bullæ or vesicles are perhaps more numerous but are smaller than those of erysipelas, and the pronounced rise of temperature and swelling of the nearest lymphatic glands which are so characteristic of erysipelas are rarely present in herpes.

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[1] Verneuil, Gazette de Paris, 1873, No. 20, *et seq.*

[2] Hutchinson, Ophth. Hosp. Rep., vol. v. p. 207, and vol. vi., February, 1869.

Hence the mistake between the two diseases ought not to occur nearly so frequently as is the case, and must arise either from carelessness or from ignorance of the facts about herpes ophthalmicus.

It should be noted that the latter have been known to ophthalmic surgeons only since 1866, that it is an uncommon disease, and that many medical men have escaped seeing or hearing of a case. There is also an important point to be noted,—viz., that an attack of herpes may be complicated after a time by genuine erysipelas, though this is far from frequent.

In the diagnosis between the two, the chief points are the peculiar distribution of the cutaneous inflammation in herpes and the fact that it does not travel when once developed.

Further, the pain preceding the outbreak is more prolonged and more intense in herpes than in erysipelas, though to this rule there are exceptions.

The congestion of the whole eye and the pain in herpes may suggest acute glaucoma, and some increase of tension may be present with the former, but the distribution of the vesicles, etc., will usually prevent such a mistake in diagnosis.

It is unnecessary to discuss the diagnosis of herpes from so-called pemphigus or essential shrinking of the conjunctiva, since there is little probability of the two diseases being confounded.

There is, however, another form of herpes which requires to be distinguished from the one already considered.

Herpes of the eyelids and neighboring parts may not be due to neuritis of the sensory nerves, but induced and localized by sympathetic nerve irritation. In other words, febrile herpes, with its initial rigor, may (rarely, it is true) affect other parts of the face than the region of the lips, where it is so well known. This is an important fact, since it explains the anomalous cases of supposed herpes zoster affecting both sides of the face. A good example is reported under the heading of severe bilateral herpes ophthalmicus by Dr. W. Robertson.[1] In this case the vesicles, bullæ, and pustules occurred in a circle around each eye, and were supposed to be in the distribution of the frontal, nasal, infra-orbital, and lacrymal nerves. The illustration accompanying the report enables one to see that they were exactly symmetrical, did not affect the whole area of any one of these sensory nerves (for instance, above the eyebrow the vesicles extended for only one inch,—a limit never respected by true herpes frontalis), and were more probably determined by the sympathetic nerve reacting on both facial arteries. The patient was suffering from pneumonia and nephritis at the time of the herpetic outbreak.

We may grant that herpes in the distribution of sensory nerves is occasionally symmetrical, but it is open to doubt if any true bilateral case has been yet observed in the face, and the above example illustrates a fallacy which has to be guarded against.

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[1] Lancet, July 7, 1888.

With regard to the *prognosis* of herpes ophthalmicus, the severity both of the eye-lesions and of the skin-eruption is apt to be greatest in patients of advanced age. Not only does the pain persist in some cases for long periods, but the affected eye may be left irritable and with increased tension. "In almost all cases the tract of skin involved in the eruption remains for long partially anæsthetic. When the eye has been involved, the cornea frequently loses sensation, and never fully regains it."[1] This fact must render it especially liable to inflammatory trouble subsequently, from entrance of dust or other foreign body. Then, again, the prognosis must be grave should iritis develop with the herpes.

*Treatment.*—If the pain is severe, hypodermic injections of morphine or the internal administration of quinine and opium may be advisable. Leloir recommends the local use of an alcoholic solution of cocaine hydrochlorate or of menthol (fifteen grains to the ounce of either) to the affected area of skin. The physician should carefully abstain from using (as has been recommended) a strong solution of nitrate of silver or other caustic, since serious harm (acute cellulitis, or even gangrene) has been known to follow. The mildest treatment is alone required, but, since there is slight danger of erysipelas or septic infection occurring, it is wise to use an antiseptic application. Probably nothing is better than a warm fomentation of boracic lint soaked in weak carbolic solution. The use of subacetate of lead lotion should be avoided, since if there be any corneal lesion a deposit of the oxide may occur.

In the event of iritis supervening of course atropine must be applied, and, perhaps owing to the conjunctival congestion, it is particularly difficult to obtain proper mydriatic effects from it: hence a very frequent application of a strong solution is advisable, as soon as the pupil is noted to act sluggishly, until free dilatation is procured. Since many of the cases are in old subjects, in whom atropine is apt to bring on glaucoma, cocaine should be used with the other alkaloid.

The patient should be kept in bed, or, at any rate, protected from draughts, while the attack is at its height. A nourishing diet, quinine and port wine, etc., are to be recommended in most cases; and if the neuralgia prove intractable, a surgical operation (division of the supra-orbital nerve after Thiersch's method, with extraction of a long piece of the trunk) may be required.

In conclusion, stress may again be laid on the facts that herpes ophthalmicus is the frequent subject of mistakes in diagnosis, and that if the eye is markedly affected the prognosis must be very guarded. Vision may be wholly lost, owing to iritis or destruction of the cornea, or, in less grave cases, the eye may be left irritable and congested or with increased tension long after the attack of herpes has subsided.

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[1] Hutchinson, Royal London Ophthalmic Hospital Reports.

# MOTOR CHANGES IN THE OCULAR APPARATUS ASSOCIATED WITH FUNCTIONAL NEUROSES.

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BINOCULAR single vision implies the perfect correlation of many factors, both physical and psychical. The retina of each eye receives a picture which is carried by the nerve to the percipient centres. These pictures, although not identical, are mentally combined and recognized as representing one object. This result is accomplished only, in all probability, when the percipient centres have come to recognize the fact that the vision of an object falling upon the macula of each eye must be mentally interpreted as one object, and not as two. In course of time this knowledge becomes so automatic that if for any reason the picture of the object looked at falls in one eye elsewhere than on the macula, two images are immediately perceived and confusion results. In order that this parallelism of the visual axes of the eyes shall be maintained with as little muscular and nervous fatigue as possible, it is necessary that it should always be present without active, conscious or unconscious effort. The importance of this fact has long been recognized, and a weakness of the adducting muscles has been for many years considered a defect which produced asthenopia and ocular strain. The probability that such a defect or that other inequalities of ocular balance might be a source of nervous irritation sufficient to precipitate or produce grave functional nervous disturbances other than asthenopia and headache was first brought before the profession by Dr. George T. Stevens, when his successful prize essay was presented to the Académie Royale de Médecine of Belgium in 1883. Since his first advocacy of this theory, Dr. Stevens has not ceased to urge the importance of this subject upon the medical profession, and to suggest methods for the discovery and diagnosis of such errors, as well as to perfect methods for their relief when found. His views have by no means been fully accepted by the profession at large, and have been bitterly opposed by some, who have even denied that any nervous strain could arise from inequalities of the balance of the ocular muscles.

That the external ocular muscles are often so placed that a subconscious effort is required in order to avoid diplopia and its uncomfortable results will be disputed by none. Sometimes the amount of muscular effort necessary to accomplish this is so great that the habit is never acquired and the psychology of vision is much changed, as, for instance, in alternating strabismus, where the image received by the fixing eye is alone perceived, and the image of the other eye is ignored to such an extent that it cannot be discovered by any effort of the will; yet when the fixation is changed to the second eye and the second image is recognized by the consciousness, the image received by the first eye is equally ignored, and, in its turn, cannot be recalled by any effort of the will as long as the second eye remains the fixing eye. When, however, binocular vision has been established, the muscular balance must be such that not only parallelism of the axes of vision shall be maintained while looking at distant objects, but also convergence must be easily maintained when looking at near objects.

Clearness of vision requires that the rays of light which form the image on the retina shall be accurately focussed, and this result necessitates that there shall be exercised a suitable amount of accommodation for every given amount of convergence upon looking from a distant to a near object. An error of refraction may blur the picture for a near as well as for a distant object, but whenever it is possible so to do, the eye brings to its aid the ciliary muscle and by this means corrects the error of focus and secures a sharp definition. Now, it is an established fact in ophthalmology that headache, mental, physical, and nervous fatigue, amounting even to confusion and inability to think, may be dependent upon this correction of an error of refraction by the ciliary muscle. These symptoms must be due either to fatigue of the muscular apparatus, or to nervous fatigue of the central system developed by a constant semi-conscious or unconscious effort to hold the ciliary muscle up to its duty, or, it may be, to mental fatigue of the percipient centres due to the difficulty of recognizing a partially blurred image; yet it is to be remembered that for every effort of accommodation there is associated convergence, requiring also nervous stimulus, and that these two separate acts are so associated, either from actual correlation of the brain-cells or from habit, that it is only with great difficulty possible to disassociate the two acts and not accommodate upon convergence or converge upon accommodation, if indeed the latter is ever accomplished by one whose eyes have both normal refraction and normal muscular conditions.

The third cranial nerve supplies not only the ciliary muscle and the sphincter of the iris, but also the internal rectus, superior rectus, inferior rectus, and superior oblique, from which anatomical arrangement it is evident that accommodation and convergence are necessarily associated and of equal importance for comfortable binocular single vision. It would seem, therefore, that if an error of refraction can produce headache and mental, physical, or nervous fatigue, the same symptoms might be equally due to a

weak convergence. The same stimulus proceeding from the central nervous system must be present in urging the recti muscles up to their duty as is present in urging the ciliary muscles up to their duty. That mental and physical fatigue, accompanied by nervous disturbances, may be due to refractive errors or to want of perfect muscular coördination is easily demonstrated by the promptness with which such phenomena disappear when near work is entirely omitted, and also by the equally striking correlative fact that no amount of rest prevents a return of the nervous phenomena upon the resumption of near work under similar circumstances.

It is a well-known fact that some forms of refractive strain give rise to nervous phenomena more frequently than others, and this is especially true when much near work is done by the patient.

Hypermetropia and hypermetropic astigmatism, especially when the axes are oblique, may cause nervous irritation sufficient to be a source of almost constant headache and other nervous phenomena; yet if the refractive error is properly corrected and glasses are constantly worn, the nervous disturbance ceases. In these cases the nervous irritation must be very largely dependent upon fatigue of the ciliary muscles and upon the necessity of continued nervous stimulation to keep them up to their work. If this is so true as to be almost an axiom in ophthalmology, must it not also be true that if the opposing external ocular muscles are of unequal strength, or are so inserted into the sclera as to have an unequal purchase, the effort to maintain the proper amount of parallelism and convergence to avoid diplopia must excite a fatigue and irritation of the nervous system similar and equal to those which arise from refractive inequalities?

In errors of refraction one may have excessive nervous disturbance from a small error on account of the nervous susceptibility of the patient to this form of strain, or, on the other hand, one may have large errors of refraction and no nervous disturbance resulting therefrom. So in the muscular errors, a large amount of nervous disturbance may arise from small errors, and some of the most brilliant results which I have seen after graduated tenotomies have been in cases where the error was not great.

An attempt to define the normal muscular balance is somewhat difficult, but in my opinion the normal muscular condition is such that, when looking at infinity, there should be an absolute esophoria of one or two degrees, and that upon converging to fifteen inches a normal muscular balance should exhibit an exophoria of about three degrees. Vertically, the condition of orthophoria seems to be the proper muscular balance, always bearing in mind that when an orthophoria exists vertically such a plane of equilibrium should be at or below the horizon in order to give comfort in most of the occupations of life.

The above condition defined as normal applies only to eyes with normal refraction, or at least with but moderate error.

In high hypermetropia, myopia, or high astigmatism, either myopic or hypermetropic, comfortable vision may require a muscular balance varying

greatly from the above standard. In these cases when the refractive error is corrected a prismatic element placed in the glass is much more apt to give permanent relief than when such a correction with prisms is attempted with lower errors of refraction.

It is often the case that there may be no complaint from the nerves until there is long-continued application to work which requires exact fixation during many hours of the day. Indeed, in an ocular strain, refractive or muscular, nervous disturbance is not directly dependent upon the amount of error, or upon its kind, for muscular and refractive errors of both large and small amounts are quite common in cases in which there is no nervous strain whatever; and again, the amount of a muscular or refractive error is no indication of the severity of the resulting nervous disturbance. The reason of this undoubtedly is that to have a muscular or refractive error produce a nervous explosion of any sort, a neurotic inheritance, or at least an unstable nervous equilibrium, must exist in the patient, and when to this predisposition to migraine, neurasthenia, chorea, or epilepsy there is added an ocular strain, it becomes the precipitating irritation which calls forth a group of nervous symptoms, while it is not, in a strict sense of the word, the cause of the nervous disturbance. Before considering the subjects of epilepsy, chorea, and migraine, I wish to enumerate the symptoms which are most commonly present in neurasthenical cases in which the abnormalities of muscular balance are the most pronounced ocular errors.

## NEURASTHENIA.

Such cases complain of pain in the occipital region, often of a pain in the back of the neck, not infrequently of pain in the spine, down as far as between the scapulæ; often of a giddiness, which is never so pronounced that the patient falls, and is most frequently present upon first arising in the morning, in crowded streets, or when looking at rapidly moving objects. They are very apt to complain of a sense of confusion in thronged thoroughfares; of being car-sick on railway journeys; of being unable to watch the passing scenery from a railroad train without fatigue, headache, and, perhaps, confusion; that they are unable to study, or to concentrate their thoughts upon the subject-matter of the text, or to think logically in connection with what they read; that they do not like to meet and talk to people or look steadily at any object. If clergymen or public speakers, they are unable to speak effectively because of a mental confusion which seizes them upon looking alternately from the manuscript to the audience. These symptoms, when the occupation of the individual is such as to call them forth, slowly increase in intensity, and gradually the headaches are not confined to the occipital region, but become more and more a marked feature of the clinical picture, until, finally, the patient is reduced to a condition of neurasthenia, which compels him to abandon his occupation, retire to some quiet locality, and spend months in allowing his mental and nervous equilibrium to re-establish itself. Then only too often upon re-

turning to his usual work the same story repeats itself again and again, until the patient finally becomes a nervous, querulous, irritable invalid, utterly unfitted for any occupation which requires either the use of the eyes or mental application. That such an apparently hopeless condition can be relieved with promptness and permanently by a graduated tenotomy of the overbalancing muscle or an advancement of the weak muscle is a fact that has been established to my satisfaction by a number of cases in my own practice. I have selected two of these cases to report here which are sufficiently typical to illustrate this form of neurasthenia.

CASE I.—A lawyer forty years of age had been unable to work more than an hour a day for thirteen years on account of pain in the eyes for all near work, which increased to dizziness upon looking up from work. He had also severe headaches, the pain extending from the occiput down the spine. His health was otherwise fairly good, except for a tendency to dyspepsia. He had had glasses prescribed, after an examination under atropine, at various times without any benefit as regarded his ability to attend to his affairs. He had worn glasses more or less since 1877. He was first seen by me September 14, 1889. He had then manifest hypermetropia of 0.75 D. in each eye,—V. o. u. = 20 / XX. After a careful examination, running through several weeks, I determined that he had an esophoria of 4° and no hyperphoria. On October 14, 1889, I did a graduated tenotomy of the internal rectus of the left eye, which left, as a final result, on the 29th of the same month, less than one degree of exophoria, being practically orthophoria. There was immediate improvement, and the following January the patient was doing a full day's work. In September, 1892, he reported himself as perfectly well up to that time, using his eyes all day at the registry of deeds. He was last seen by me November 18, 1895, when he was still well, and a trial of the muscular balance showed a state of orthophoria.

CASE II.—A clergyman thirty-one years of age. First seen by me August 11, 1887. He had had much asthenopia for six or seven years. He had been examined by several gentlemen distinguished in the profession; had glasses prescribed; and had tried Dyer's method for the progressive use of the eyes. The last two years the headache from the use of the eyes had increased so that he had been obliged to give up all reading, and the least excitement prostrated him. He could not go down town in the street-cars, or make a social call, except in the dark, without precipitating a violent headache. He had been obliged to give up his duties on account of his infirmity, and for ten months had been almost an invalid. He could not read more than two pages without being obliged to stop on account of pain. V. o. u. = 12 / X; exophoria, 4° to 5°. The patient was examined frequently during the following nine months, and the amount of exophoria was found to be constant. On May 21, 1888, I did a graduated tenotomy of the internal rectus of the left eye, leaving something less than one degree of exophoria. There was prompt relief, and in July of the

same year he returned to his clerical duties, accomplishing them without fatigue. From that time he has been able to do continuous work. He was last seen by me May 25, 1897, when he said that the constant pain in the head and back which was relieved by the operation had never returned,—that, while he had to be somewhat cautious in the use of his eyes, he had been able to do all the work necessary for his position and had enjoyed good health. A trial of the muscular balance showed less than one degree of exophoria.

In these two cases it will be seen that the patients were permanently relieved of a condition of neurasthenia by the operation; it will also be noticed that the operation gave an absolute decrease of the amount of heterophoria, and it is of further interest to note that the muscular balance which obtained after the operation has been absolutely permanent after an interval of six and nine years respectively.

The great stumbling-block in the way of the successful treatment of these cases is the difficulty of the diagnosis. Outbreaks of functional nervous disturbance may be dependent upon the strain caused by an error of refraction, the strain caused by abnormalities of the muscular balance of the external ocular muscles, or a strain arising from both sources. Moreover, the interdependence of accommodation and convergence may create a false muscular action, which is entirely dependent upon the amount of nervous energy required to correct a distressing error of refraction, and it is equally true that the constant effort required to overcome insufficiencies of the external ocular muscles may cause functional astigmatism. This being so, before any opinion can be given as to the existence or non-existence of a proper muscular equilibrium, it is necessary that an examination of the refraction should be made under a mydriatic, and proper lenses worn to correct the refractive errors for a sufficient time to allow all spasmodic and compensatory muscular action to subside. Even then the muscular balance must be measured a number of times under varying conditions, with and without the lenses, before we can be sure that the apparent errors of muscular equilibrium are the actual dynamic conditions.

It has been my fortune many times to see an apparent muscular error utterly disappear after proper correction of the refraction. It is not the purpose of this paper to describe the methods of examination for muscular errors, as this subject has been treated by another contributor to the System, who is better fitted to teach them.

In all cases of muscular anomalies it is of importance that we accurately determine whether the lack of equilibrium is not due to over-stimulation of some one or more muscles. This over-stimulation of an ocular muscle may arise from an irritation of the nerve governing the muscle or in the early stages of degenerative diseases of the brain or spinal cord. Dr. E. D. Spear has called attention to the fact that in many cases of disease of the middle ear the transmission of pressure to the semicircular canals of the labyrinth may set up an irritative over-stimulation of the sixth nerve, and perhaps

the third, and so produce such an overaction of one or more of the external ocular muscles that there will be great complaint of dizziness and an inability to walk in a straight line, and that the prism tests will then show an apparent exophoria or hyperphoria; yet relief of the intra-aural tension will immediately cure the dizziness, and the apparent exophoria or hyperphoria will instantly disappear. This fact is well illustrated in a case of my own. A man fifty-six years of age, who had always had good eyes and been well, came to me reporting that he had had in the past year several dizzy spells, and that six days before his visit to me he had several vertiginous attacks in one day, and the next morning found that he could not walk with safety. This condition continued until his visit to my office. I found that on placing a red glass before one eye the candle test gave immediate diplopia, and that the eye turned up and out. A prism of eight degrees was required over each eye to do away with the diplopia. I referred the gentleman to Dr. Spear, who reported that he found great swelling of the right turbinated body, a closed Eustachian tube, depressed cicatricial membrana tympani with fair hearing, and that treatment of the Eustachian tube immediately did away with the dizziness. The eye returned to its normal position and the patient could walk in a straight line.

The route of the transfer of nervous irritation from the ear to the eye is probably through a connection between the eighth cranial nerve and the sixth, which is brought about in the following way. The cochlear branch of the eighth nerve enters the ventral nucleus, whence fibres forming the trapezoid body are thrown off. A certain number of the trapezoid fibres lead directly to the superior olive. From the superior olive a well-marked tract takes a course towards the fourth ventricle and enters into association with the nucleus of the sixth nerve, thereby effecting a connection between the special nerve of hearing (cochlea) and the abducens supplying the external rectus of the eye. The purpose of this connection is undoubtedly to aid in maintaining the equilibrium. This connection is well shown in the accompanying diagram (Fig. 1), drawn by Dr. E. W. Taylor, after a plate by Edinger.

It is, therefore, absolutely necessary to determine whether a muscular condition is anatomical or pathological, for if the trouble is due to overstimulation of the muscles from pathological conditions, it is obvious that operative interference is improper, as in cases similar to the one reported above it is evident that upon the re-establishment of more normal aural conditions the result of an operation would be disastrous. When the irritation proceeds from degenerative disease of the brain or spinal cord, an operation gives very little benefit, for the continuance of the nervous irritation soon reproduces the same condition that existed before the operation.

This last phenomenon is not to be confounded with that re-establishment of a muscular error after a graduated tenotomy which is due to the development of a latent insufficiency. This it is which so often makes it necessary to do repeated operations in these cases, a fact which has brought

opprobrium on this method of procedure. If, however, the latent insufficiency is developed as much as possible by the use of prisms immediately

Fig. 1.

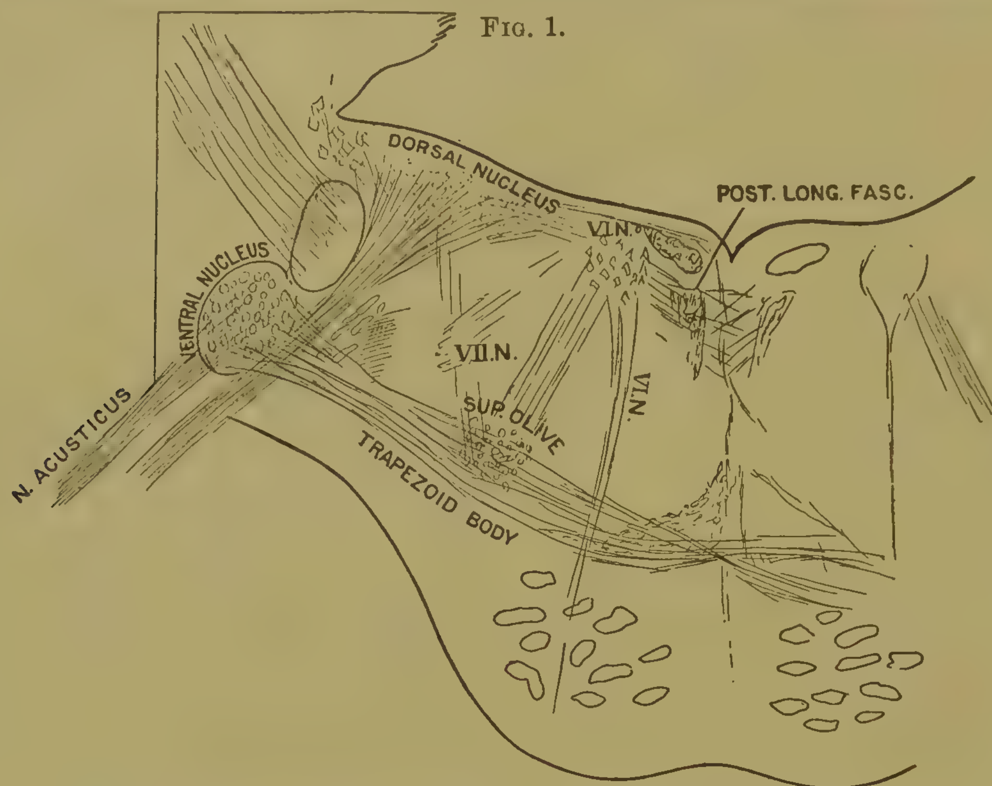


Diagram illustrating the connection between the eighth and sixth cranial nerves.

before the operation, we can graduate the amount of operative procedure so that supplementary operations can be to a great extent avoided.

## EPILEPSY.

Epilepsy may be regarded as a symptom-complex which occurs in persons predisposed thereto from a great variety of causes. Many of these are organic, as, for instance, depressed fractures of the cranial bones, tumors of the brain, syphilis, meningitis, abscess of the brain, peripheral nerve diseases, or anatomical, as in porencephalic brains. In all these instances it is evidently purely a symptom. The same symptom-complex, however, may be precipitated by purely functional causes in persons predisposed thereto, —mental or gastric disturbances, fatigue or excitement,—and in these cases it is also only a symptom indicating a tendency towards this form of nervous excitability. In all probability the so-called idiopathic epilepsy is also purely a symptom-complex existing in persons predisposed thereto and precipitated by some functional reflex disturbance which is undiscovered, and probably as yet unsuspected.

It is undoubtedly true that the symptom-complex which we speak of as epilepsy may have its starting-point in ocular strain, and may be prevented by the correction of the error even when this error is simply refractive, as the following case will show. Mary H., thirteen years of age, had the following family history. An aunt on her mother's side had chorea. The father's mother was insane. A sister of the patient had had "fainting fits" for a long time. This sister had an error of refraction, but did not wear

glasses. The patient had always been well up to her eighth year, when she had epileptic convulsions preceded by a cry, after which she became rigid and had general convulsive movements. She would remain unconscious for some moments, and after recovery from the attacks was sleepy. She was much more subject to these attacks when she attended school, and they had continued with more or less frequency for five years. She had a marked hypermetropic astigmatism, for which glasses were prescribed. She had one slight convulsion on the night following the first day of wearing glasses, after which there were no more epileptic convulsions. If, then, this distressing neurotic condition can be prevented by the correction of a refractive error, there is no reason, as has been urged before, why an ocular strain coming from a difficulty in maintaining binocular vision should not also precipitate, in cases predisposed thereto, epileptic convulsions. It would seem also to be well established as a matter of fact, as well as from analogy in other functional nervous disturbances, that the symptom-complex called epilepsy soon tends to become a fixed habit, and the more the brain has been subjected to periodic discharges of nervous force of this sort the more prone it becomes to repeated attacks. Not only this, but the tendency is for the attacks to become more and more severe as time goes by, and it is not impossible that in persons of neurotic family history and of great nervous excitability themselves a habit of this sort, which at first might have been discontinued if the particular source of nervous irritation had been early removed, may in time become so fixed and overpowering that it may continue after the original cause is removed, and the attacks then be precipitated by many other sources of reflex nervous irritation. If this is true, it is clear that the earlier the source of a reflex nervous irritation which is producing epileptic convulsion is removed the greater is the chance of a radical cure.

Of course no one will argue that an epilepsy due to an abscess of the brain or to syphilis could be cured by removing the ocular strain if present. Neither would any one argue that epileptic convulsions due to a chronic meningitis could be cured by the removal of any source of ocular strain. The most that could be expected from an operation in such a case would be that the fatigue from the use of the eyes for near work should not thereafter be a direct cause of an epileptic attack. Whether it would be advisable to operate would, of course, depend entirely upon the history, which might show that the convulsions frequently followed near work. When the epileptic tendency exists without any organic change in the brain, it is evident that any reflex irritation may be a precipitating cause of the nerve habit. Under such conditions ocular strain is one of the many sources of irritation, and if the principal source, an operation which does away with the fatigue will undoubtedly be of much benefit to the patient. The amount of relief from the epileptic attacks which may be derived from doing away with ocular strain, when that is the precipitating cause, will, of course, vary in proportion to the length of time that the patient has been subject

to such nervous crises, and the most brilliant results will be obtained in cases in which the epileptic habit has not been of long duration.

In my personal experience the most successful cases are among children and young adults who are still students, or whose occupations are such as to make great demands upon the eyes at the time when the first epileptic convulsion occurred. A case in point is that of a boy eleven years of age, who was brought to me on September 24, 1889. He had epileptic convulsions, the first of which had occurred a year before his visit to me. At this time he had two attacks with a short interval between them. He had no more until March, 1889, when a third convulsion occurred. This one had been followed by a number at varying intervals in the succeeding six months. He complained of a slight headache much of the time. Said his eyes ached sometimes. Had taken bromide of potassium regularly the last six months. V. o. s. = 20 / XV + 5. V. o. d. = 20 / XV + 4. An exophoria of 3° was manifest. No hyperphoria. On September 30 an examination of the refraction under atropine showed H. o. u. = .50 D., exophoria 4°. Glasses were prescribed for diagnostic purposes, correcting the error of refraction, and each lens contained a 2° prism, the base of which was placed towards the median line. These were worn until November 4, when he reported that he had been quite well in the interval, except for a slight headache at times. Examination showed an exophoria of 7°. The lenses were then reversed, and the bases of the prisms placed outward. On November 14 it was reported that he had been much more restless, had suffered much more from headache, and had had several epileptic convulsions. The manifest exophoria was then only 1°, due to the stimulation of the weak muscles by the burden placed on them by the arrangement of the prisms. On January 18, 1890, I did a graduated tenotomy of the external rectus of the right eye, leaving only the last few fibres on each side of the tendon. The immediate result of the operation was orthophoria at twenty feet. On December 4 of the same year the mother reported to me that the boy appeared much brighter since the operation, and that the epileptic manifestations had been confined to two attacks of petit mal, one of which occurred after a fatiguing day in the city and the other after watching a procession for eight hours. On July 1, 1897, in response to a written inquiry, I was informed that the young man was in perfect health and had completed his second year in college. He had had no epileptic manifestations since the two attacks of petit mal above reported, and had worn no glasses since the operation.

## CHOREA.

As with epilepsy, so chorea may also be regarded as a symptom, a motor disturbance, which is characterized by physical movements, indicating an irritative nervous process. Choreic movements may be due to organic disease, as, for instance, post-hemiplegic chorea, and perhaps the so-called Huntingdon's chorea, which is an hereditary disease and goes on to mental disturbance and death; but in a large proportion of all choreic

cases the choreic movements are only a symptom which is produced in persons predisposed thereto by some irritative nervous condition. Many cases have a history of rheumatism and endocarditis, and it has been argued that a condition arising from rheumatism, then having endocarditis, and after that chorea, was due to a toxic condition resulting from rheumatism, or that all three conditions came from some antecedent toxic source. If either of these suppositions were true, it does not place chorea anywhere except as a symptom. A large proportion of choreas, however, happen in childhood, when nervous disturbances are easily precipitated, and very often commence in the early spring months, when children have been in school all winter and often studying by artificial light. These cases upon being removed from school recover during the long summer vacation, and the choreic movements reappear only after they have returned to school and again used their eyes for study some months. In my experience, such cases are more apt to be due to errors of refraction than to muscular anomalies, and children who have been removed from school two, three, or four successive years in the early spring months will often have no return of the symptom if proper glasses are used. The following case is a good example. Minnie F., seventeen years of age, had the following family history. One aunt on the mother's side had chorea. A brother thirteen years old has had four attacks of chorea, the first when he was seven years old. The patient is the third child of five, and has had rheumatism. In January, four years before the first visit to the hospital, she developed chorea of such a severe type that she could not sit in a chair, or feed or dress herself without aid. She was removed from school, sent into the country, and returned at the end of ten weeks well. In January of the following year she had a second attack, lasting between three and four months. The movements at this time were not as great as those of the previous year. The following December the condition returned in as severe a form as when it first appeared. All three attacks were preceded for some weeks by twitching of the muscles of the face, especially about the eyes. Physical examination showed slight mitral regurgitation. She had taken Fowler's solution, but had ceased four weeks before her visit to the hospital. The choreic condition was well marked; there were violent twitchings of both upper and lower extremities. Upon examination, hypermetropic astigmatism was found, and glasses were prescribed. There was an immediate cure, and there has been no relapse. When correcting the error of refraction does not prevent a return of choreic movements and when muscular anomalies are present, it would seem logical that correction of these conditions might be necessary in order to prevent a return of the choreic condition, and this supposition proves to be a fact upon trial, as the following case, reported by Dr. F. E. Cheney, will show. A boy eight years of age was first seen in the ophthalmological out-patient department of the Massachusetts General Hospital, July, 1889. The mother of the child reported that the chorea began during his fifth year, and although the twitchings were at times very slight, he had never since been entirely

free from them and was constantly subject to violent attacks, which were much more frequent when he was attending school. He had never complained of pain in the eyes or of dizziness. Within the last few months a slight convergence of the left eye had been noticed. The patient was first seen at the neurological out-patient department of the same hospital, October, 1887, five months after the commencement of the chorea. He was at that time under treatment nearly three months, when he was reported to have been nearly well. A few months later he had a second attack, succeeded by others. At the time of his visit in 1889 the choreic movements were said to have been of considerable severity for more than two months, the twitchings involving the muscles of both upper and lower extremities, neck, and face. Examination under atropine showed the following conditions. Hypermetropia 1.50 D. o. u. Also that when a colored glass was placed before one eye, diplopia ensued, and the left eye converged slightly. There was an esophoria of 8°. Glasses were prescribed for constant use. The correction of the refraction produced little, if any, change in the patient's condition. An operation for the correction of the muscular anomaly seemed indicated, and Dr. Cheney, on August 8, did a complete tenotomy of the left rectus internus. The patient returned five days later decidedly better. The twitchings were not severe and were far less frequent. August 20 the twitchings had nearly ceased. October 1 there were no choreic movements; the boy was attending school and perfectly well in every respect.

In the symptom-complex called chorea, as in epilepsy, the amount of relief which may be obtained from doing away with the ocular strain will depend upon how long the condition has continued and how fixed the brain habit has become; but I have seen young people pursue an educative course of considerable difficulty after relief of muscular anomalies when all previous efforts by general treatment—physical care, mental quiet, or correction of the errors of refraction—had not prevented a return of the choreic movements whenever studies had been resumed.

## MIGRAINE.

As epilepsy may be regarded as a nervous disturbance which expresses itself through the irritation of the motor centres, so migraine may be regarded as a nervous disturbance which expresses itself through the irritation of the sensory centres. An attack of migraine, as a rule, commences with waves before the eyes, with perhaps an apparent rotation of circular objects, or a lateral movement of vertical lines. These symptoms are often followed by temporary loss of the whole or a portion of the field of vision, accompanied by dizziness and nausea; this in turn is succeeded by a violent headache, often lasting for hours. That migraine is closely allied to epileptic convulsions would seem to be acknowledged. As in epilepsy, so here, various causes may precipitate such a nerve-storm,—excitement, general bodily fatigue, emotional experiences, and, last but not least, ocular fatigue. There would seem to be no reason why attacks of migraine

should not be precipitated by the same ocular strains that precipitate epileptic convulsions, but my own experience leads me to think that refractive strain is a much more fruitful cause of attacks of migraine than abnormalities of the ocular muscles.

In regard to the treatment of any functional nervous disturbance by operation upon the ocular muscles, it is evident that success is absolutely dependent upon an accurate diagnosis, and we must satisfy ourselves in the presence of neurasthenia, epilepsy, chorea, or migraine, not only that there is a lack of balance of the ocular muscles, but that the muscular error is a direct precipitating cause of these attacks. No one will dispute that this accuracy, so much to be desired, is a very difficult matter; but a prognosis as to the benefit to be derived from an operation can generally be made with considerable certainty if prisms which relieve the overburdened muscle give relief to the nervous symptoms, and more especially if prisms which add to the load of the already overburdened muscle increase the nervous symptoms. This method of diagnosis at times, however, utterly fails, as in certain cases prisms placed in either direction will give a temporary relief from the symptoms, due in all probability to the fact that the combination which has produced the fatigue is destroyed, and the burden is put on a new place which must in its turn become fatigued before the nervous irritability is again reproduced.

All cases of functional nervous disturbance with anomalies of the muscular balance require patient study and the use of many expedients, but even with all the methods of investigation at our command at the present time absolute accuracy of diagnosis is in many cases unattainable.

## THE OPERATION OF GRADUATED TENOTOMY.

The technique of graduated tenotomies is not difficult, provided the instruments devised by Dr. G. T. Stevens are used. The ordinary strabismus hooks and scissors are too clumsy for this delicate operation.

The steps of the operation as devised by Dr. Stevens are as follows. The speculum having been inserted and allowed to spring open only enough to hold it safely in place, the patient is then directed to fix the vision steadily upon some designated point. This point is selected so as to place the muscle to be operated upon on the stretch and to bring the seat of the operation, where the tendon of the muscle is inserted into the sclera, clearly into view. The operator then takes his fine forceps (Fig. 2) and seizes a small fold of the conjunctiva directly over the centre of the insertion of the tendon. Drawing the little fold of conjunctiva slightly away from the eyeball, he snips the fold transversely with his fine-pointed tenotomy scissors (Fig. 3), so that an opening of one millimetre is made through the membrane. The forceps are then closed, pressed into the little opening, and allowed to spring apart, after which they are again closed, this time grasping a small fold of the tendon just behind the insertion. This little fold of tendon is then put on the stretch, and a small opening is made in

the centre with the fine-pointed scissors. One blade of the scissors is then inserted beneath the tendon, which is then dissected from the eyeball by little snips proceeding towards one edge of the tendon as far as the operator deems sufficient to meet the requirements of the case. Then, turning the scissors in the direction of the other border, the procedure is repeated in that direction.

FIG. 2.

Fine-pointed tenotomy forceps.

FIG. 3.

Narrow-pointed tenotomy scissors.

The speculum is then removed and the condition of the ocular balance is tested. If any considerable amount of the original error remains, the speculum is replaced and the incision in the insertion of the tendon extended a minute amount. This is done by inserting the slender hook (Fig. 4) and

FIG. 4.

Slender tenotomy hook.

determining the point to which the dissection has been carried in each direction, and if he finds room to carry the section farther in either direction, he does so, using the slender hook for a guide or returning to his forceps, after which the ocular balance is again tested. The operation in cases in which the error is not large should be continued until a condition of orthophoria is reached, if this can be done without detaching the tendon entirely; for at the moment there is likely to be a greater effect from the operation than will be found a few hours or days later. In my judgment, a tendon should never be entirely severed from its attachment unless actual strabismus is present; but if from any accident this should occur and an overcorrection result, a very delicate suture must be at once inserted into the exact centre of the free end of the tendon and carried through the cut edge of the conjunctiva on the corneal side of the wound. This suture should include as little as possible of either tissue, and the knot should be drawn with great care until the operator has produced a condition of orthophoria.

The principal difficulty is that there is often considerable hemorrhage which covers the field of the operation; when this happens the opening in the conjunctiva will have to be somewhat enlarged and the continuance of the operation delayed until the bleeding has partially ceased.

It is true that the view of this question here presented is by no means universally accepted, but it is also true that a dispassionate judgment as to

the value of this theory and as to the facts presented from time to time in its substantiation has been retarded by the over-enthusiastic statements of some of its advocates.

Not only this, but the extreme laudation of the benefits to be derived from the operation of partial tenotomy has led many surgeons to take up the method with enthusiasm, and as a natural result the operation has been too indiscriminately and indiscreetly utilized. It has not been duly considered that a tenotomy, whether partial or complete, is a much more serious procedure than the prescription of lenses, and less easily remedied if the expected results are not attained. Many practitioners have also been too ready to accept a manifest muscular error as a permanent defect, and have not appreciated the necessity of a thorough study of such cases running over long periods of time, in order to establish an accurate diagnosis. Some ophthalmologists believe that whenever an error of refraction exists it should be corrected, whether it is giving rise to ocular or nervous symptoms or not, and such practitioners, whenever they have become convinced that the refractive and muscular errors were of equal importance, have attempted to correct all muscular errors wherever found, and this has resulted in much meddlesome surgery. Again, only too often, if a muscular error has been found coincident with functional nervous disturbance, it has been immediately assumed that the ocular condition was the cause of the nervous defect without any attempt to prove that the two conditions were cause and effect. The inevitable result has followed that the operation has been done when the reflex cause of the nervous disturbance has been the disarrangement of function of some other organ of the body not in any manner associated with the ocular condition. Not only this, but operations have undoubtedly been done when the ocular conditions were in themselves only a symptom, and it has been overlooked that inequalities of muscular balance may be due simply to disturbances of the nerve-supply of the affected muscles, as in the early stages of locomotor ataxia and tumor of the brain, or in the course of a chronic meningitis.

Such errors can in most cases be avoided, even when the diagnosis of the organic nervous lesion is very difficult or impossible, by a longer study of the cases, which will show that the error is a constantly increasing one, or that it is transitory and atypical. Such errors are inherent in all new methods of procedure, and always bring discredit upon new and radical lines of treatment until experience has taught the true value of the method, and determined when it is and when it is not indicated.

No accepted surgical procedure is always successful in all hands and at all times, and no operation should be condemned because it is not invariably successful.

The chief difficulty has been that operators have not appreciated the many-sidedness of the subject and the difficulties of the diagnosis.

It is too early as yet to estimate even approximately the relative importance of ocular strain in the production of functional nervous disturb-

ances such as have been considered in this article. That the proportion is small will be conceded by all fair-minded observers. Many eminent neurologists and ophthalmologists as yet deny that chorea and epilepsy are ever precipitated by ocular strain arising from inequalities of balance of the ocular muscles, notably Dr. Weir Mitchell, of Philadelphia, who states that he has never seen such a case, although he does not go so far as to deny that such cases exist, and Dr. D. B. St. John Roosa, of New York, who is entirely incredulous, and has written vigorously in opposition to the theory.

# THE TOXIC AMBLYOPIAS.

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THE toxic amblyopias (*intoxicationsamblyopien* of the Germans) naturally include, to quote Leber's definition, all amblyopias and amauroses caused by toxic substances, the word "amblyopia" being employed with the understanding that it denotes obscurity, and the word "amaurosis" more or less complete loss of vision; for example, "tobacco-amblyopia," but "quinine-amaurosis."

**Classification.**—A chemical classification is disadvantageous because substances chemically different may produce singularly analogous ocular symptoms.[1] The arrangement of the drugs simply in alphabetical order, as recommended by Knies, has nothing to commend it. Although in certain instances a clinical distinction may be drawn between retrobulbar neuritis and intoxication-amblyopia, the latter, as Groenouw puts it, being a special form of retrobulbar neuritis, from the anatomico-pathological stand-point they cannot always be sharply separated. Indeed, Schmidt-Rimpler is unwilling to make any separation at all. Casey Wood's classification of the poisons into those that directly affect the optic nerve, those that produce other forms of optic nerve and retinal disease, and those that give rise to amblyopic symptoms unaccompanied by retinal or optic nerve lesions, is in many respects the most satisfactory one that thus far has been suggested.

Recent work with new histological methods indicates that soon we will be in position to establish a satisfactory pathological classification of the toxic amblyopias; but even then it will be unwise to dispense with the name of the drug which originates the visual defect,—*e.g.*, alcoholic amblyopia,—because this name bears the same descriptive relation to the disease it causes that, for example, "albuminuric" does to the retinitis which is often associated with nephritis.

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[1] Von Jaksch, in his comprehensive work "Die Vergiftungen," makes a most elaborate chemical classification of all of the poisons, which is of great advantage in considering the general toxic symptoms, but not of much help in an analysis of the ocular conditions. The author, in his essay on "The Toxic Amblyopias: their Classification, History, Symptoms, and Pathology," attempted an arrangement based upon prominent physiological and toxic actions of the various substances, which, while convenient in many respects, is open to the same objections that obtain in the case of chemical classifications.

## I. POISONOUS SUBSTANCES OR TOXINES LIBERATED BY THEM IN THE SYSTEM WHICH PRODUCE AMBLYOPIA BY AN ACTION ON THE GANGLION CELLS, NERVE-FIBRES, OPTIC NERVES, OR THEIR VASCULAR SUPPLY.

### AMBLYOPIA FROM ALCOHOL[1] AND TOBACCO.[2]

### (*a*) *Alcohol-Amblyopia.*

**History.**—Alcoholic amblyopia was described as early as 1751 by Boerhaave, and was the subject of a thesis by Edward Doebbelin in 1851, which contains full references to the earlier writings. Deval in his well-known "Traité de l'amaurose" devotes a chapter to the subject. Later come the papers of Pagenstecher and others, and in more recent times Uhthoff's masterly research,[3] to which special reference will be made later. Even in the earlier consideration of this subject the difficulty of separating cases of amblyopia due to alcohol from those due to tobacco has been apparent; indeed, some writers are sceptical on the subject of pure alcohol-amblyopia. While it must be admitted that in the majority of instances the best that can be said is that one or other of these poisons—alcohol or tobacco—appeared to be the predominating etiological factor, still a sufficient number of cases of apparently pure alcohol-amblyopia are on record to give the affection a distinct nosological position.[4]

**Etiology.**—Amblyopia from *acute alcoholism* is rare, but has been recorded by Deneffe, Hirschberg, and others in the form of almost complete blindness without ophthalmoscopic change. Cecil MacCoy and F. M. Michael[5] report optic neuritis followed by optic nerve atrophy after the ingestion of one hundred and twenty cubic centimetres of *wood alcohol.* In rabbits and dogs the writer was unable to produce amblyopia with optic nerve lesions by inducing acute alcoholism. Very rarely medical doses of whiskey have produced visual disturbance (Knapp). Toxic doses of *methyl alcohol,* according to Mengin, may cause rapid blindness.

Amblyopia from the *chronic abuse of alcoholic stimulants* is a much more common affection than the one just described, constituting, according to Galezowski, one per cent. of all cases of toxic amblyopia, and furnishing

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[1] Amblyopia alcoholica, amblyopia potatorum, amblyopia crapulosa, amblyopia ex abusu.

[2] Tobacco-amblyopia, nicotine-amblyopia.

[3] Graefe's Archiv, 1886, Bd. xxxii., Abth. IV., Ss. 95–108, and Ibidem, 1887, Bd. xxxiii., Abth. I., Ss. 251–318.

[4] Connor (Journal of the American Medical Association, 1890 vol. xiv. p. 217) reports instances of pure alcohol-amblyopia. Fernandez (Archives of Ophthalmology, 1892, vol. xxv. p. 529) has frequently observed alcoholic amblyopia in Cuba. Tobacco had nothing to do with this condition, because, in his experience, Havana tobacco never produces amblyopia without first causing some preceding constitutional affection.

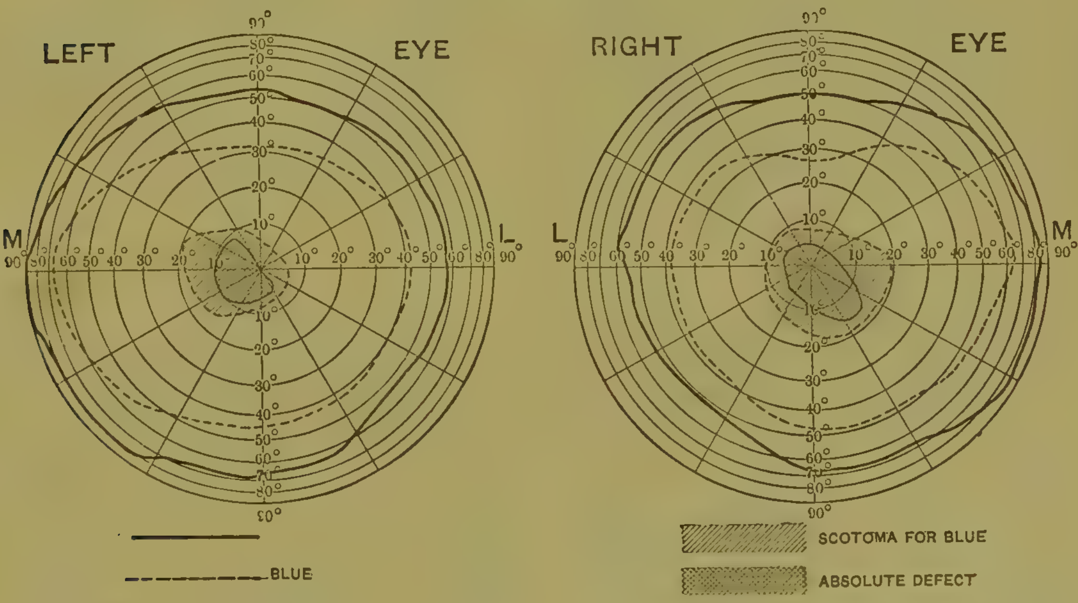
[5] New York Medical Record, 1898, vol. liii., No 22.

sixty-four of the one hundred and thirty-eight cases of toxic amblyopia analyzed by Uhthoff[1] at Schoeler's clinic in Berlin.

The amblyopia is more apt to occur when the spirituous liquors consumed contain impurities than when they are of good quality, but it is not positively known that adulterating substances, as amylic alcohol, empyreumatic oil, wormwood, oil of juniper, etc., are of themselves specially liable to create visual disturbances. Terson,[2] however, has seen over-indulgence in *absinthe* cause blindness and white atrophy of the disk in an alcoholic subject. Alcoholic amblyopia is more common among men than among women, and is rare before the thirty-fifth year.

**Symptoms.**—The usual ocular symptoms of this affection, according to Uhthoff, are: defective central vision (one-third to one-fortieth of normal), pathological whiteness of the temporal half or lower and outer quadrant of the papilla, and a central relative scotoma,—*i.e.*, a scotoma for red and green. In rare cases there is a small central scotoma for blue, and exceptionally a small absolute central scotoma surrounded by a blue-blind zone and more peripherally by a red-green-blind region. (Figs. 1 and 2.)

FIG. 1.




Alcohol-amblyopia.—Small absolute central defect, surrounded by a scotoma for blue. (Uhthoff.)

Sometimes color defects occur in the peripheral visual field. According to Demicheri,[3] these peripheral scotomas, usually sector-shaped and always relative, are found in the upper part of the visual field and are to be observed at an earlier date than the central ones. Berger[4] has found an amaurotic zone around the blind spot (amaurosis of the peri-papillary zone of the retina) in cases of alcoholic amblyopia. Very rarely there is simply

[1] Loc. cit. It should be remembered that Uhthoff classified all cases as "alcoholic" when there was evidence that alcohol was taken in excess and tobacco used only moderately.

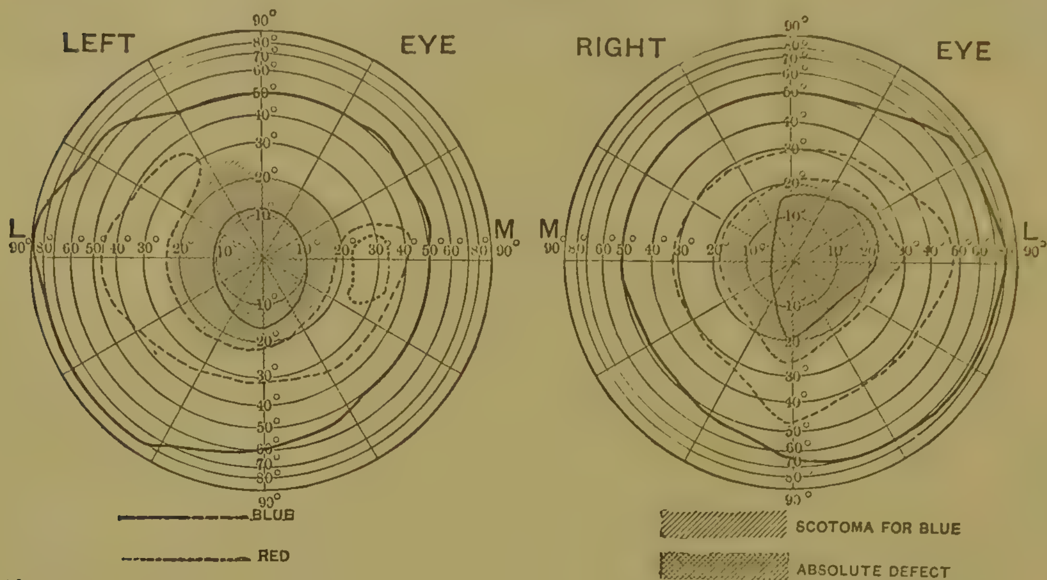
[2] Archives d'Ophtalmologie, October, 1897.

[3] Ibidem, 1896, t xvi. p. 226.

[4] Ibidem, p. 688.

contraction of the color-fields, without central scotoma. (Fig. 3.) Indeed, even when there is a central scotoma, if the field is spirally contracted,

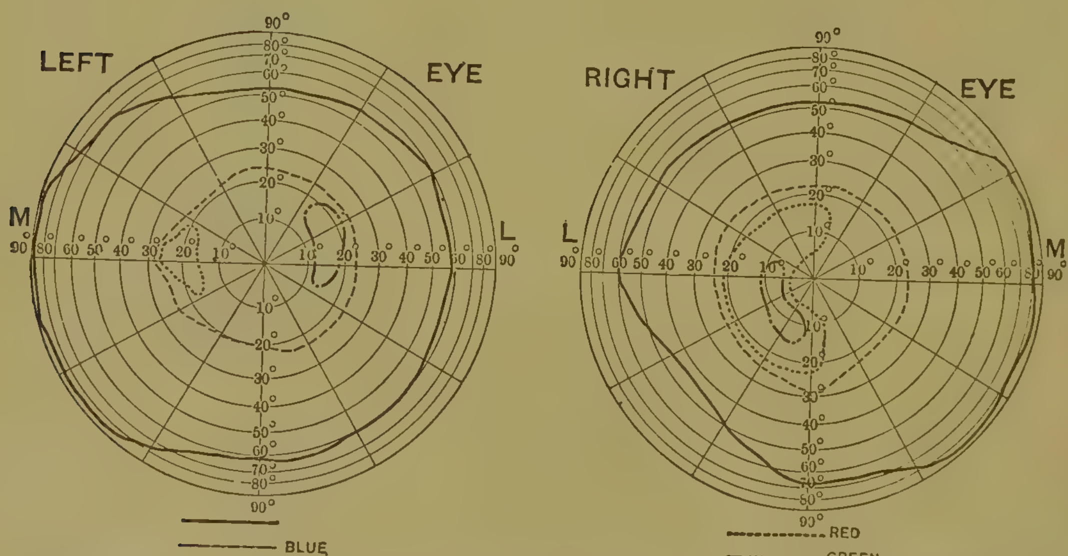
FIG. 2.




Alcohol-amblyopia.—Unusual type, illustrating large absolute central scotoma, surrounded by a scotoma for blue. (Uhthoff.)

R. Wallace Henry[1] believes it indicates that alcohol has been the predominating poisonous agent. This observation requires confirmation. Hirschberg's and Poetschke's contention that in alcohol-amblyopia the scotoma is pericentral and in tobacco-amblyopia paracentral is not correct.

FIG. 3.




Alcohol-amblyopia.—Unusual type, illustrating peripheral contraction of the color-fields, without central scotoma. (Uhthoff.)

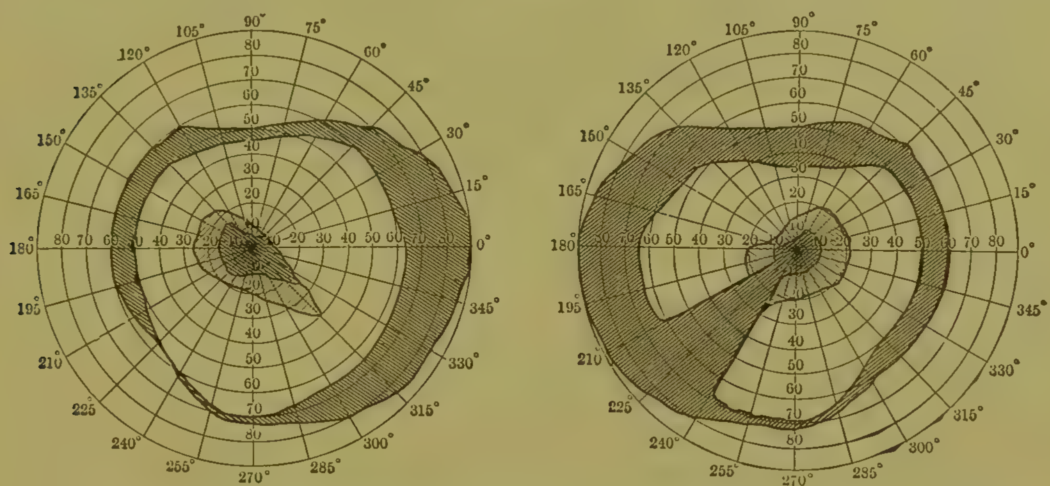
In place of the ophthalmoscopic picture usual in this and other forms of toxic amblyopia, hyperæmia of the nerve-head and even optic neuritis have been described; rarely retinal hemorrhages. These appearances are

[1] The Ophthalmic Review, February, 1896.

probably due to a slight meningitis, or convulsive seizures, which may have been present as part of the symptomatology of the chronic alcoholism from which the patient suffered.

**Prognosis.**—Under favorable circumstances,—that is, if the disease is early recognized, promptly treated, and abstinence from liquor enforced,—the prognosis is good; but there is more danger of permanent atrophy when alcohol is the sole or predominating poison than when tobacco assumes this *rôle*. Occasionally the central scotoma of alcohol-amblyopia progresses, or, rather, the affection assumes the clinical characteristics of progressive scotomatous optic nerve atrophy. (Fig. 4.) Complete blindness,

FIG. 4.



Visual fields from a case of progressive scotomatous optic nerve atrophy of toxic origin.

however, from this form, or, indeed, any form of toxic amblyopia is rare, constituting 0.039 per cent. of 2528 cases of blindness.[1]

**Pathology and Pathological Anatomy.**—This subject will be considered on page 809 in connection with the pathology of tobacco- and tobacco-alcohol-amblyopia.

### (*b*) *Tobacco-Amblyopia.*

**History.**—As early as 1792 Beer[2] appears to have been acquainted with tobacco-amblyopia, but the first scientific statement in regard to this affection originated with Mackenzie in 1832. In 1863 J. Sichel,[3] in France, and Jonathan Hutchinson,[4] in England, carefully studied the affection. A few years later appeared the communications on this subject by von Graefe, Engelhardt, Erisman, Förster, Leber, and others, in Germany, Nettleship being the first observer in England to draw accurate attention to the color scotomas.

While several observers (von Graefe, Leber, Förster) had surmised that

[1] Consult Noyes, Diseases of the Eye, second edition, p. 774.

[2] Lehre der Augenkrankheiten, Wien, 1792, zweiter Theil, S. 66.

[3] Annales d'Oculistique, 1863, t. i. p. 83.

[4] Lancet, London, 1863, vol. ii. pp. 536–538; and Medico-Chirurgical Journal, London, 1867, vol. i. pp. 411–429.

central scotoma was a symptom of lesion of the optic nerve, it remained for Samelsohn,[1] in 1880, to furnish the anatomical proof. His observations were followed by those of Nettleship and Edmunds,[2] Vossius,[3] Bunge,[4] Thomsen,[5] Uhthoff,[6] and others. While none of these papers related to tobacco-amblyopia in particular, they all furnished evidence of the anatomical situation of the lesion which causes one of its prominent symptoms,—central amblyopia.

In recent times the subject of intoxication-amblyopia with particular reference to the influence of tobacco has been investigated by Groenouw[7] and by the writer.[8]

**Etiology.**—Amblyopia from *acute tobacco-poisoning* in the form of sudden blindness has been observed a few times, for example, after application of the drug to a hollow tooth (Kosminsky), or following an enema (J. B. Wilkinson). According to the author's experiments, acute tobacco-poisoning in animals does not produce demonstrable lesions in the optic nerves. The inner layers of the retina were not examined.

Amblyopia from the chronic abuse of tobacco (*chronic tobacco-poisoning*) may be caused by smoking strong tobacco, either in pipes or cigars, by the inordinate consumption of cigarettes, by chewing tobacco,[9] and, least commonly, if at all, by taking snuff.[10] "Dipping," or the habit of rubbing snuff upon the gums with a brush or chewed stick,—a practice said to be confined to certain women of the poorer class in the south of the United States,—has been credited with producing toxic atrophy of the optic nerve by Blitz[11] and G. P. Hall.[12] Finally, amblyopia may occur in those who do not use tobacco in any form, but who work in tobacco manufactories. The author has seen one such case.[13]

The *quantity* of tobacco likely to be followed by deleterious effects on

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[1] Graefe's Archiv, 1882, Bd. xxviii., Abth. I., Ss. 1–110.

[2] Transactions of the Ophthalmological Society of the United Kingdom, 1881, vol. i. p. 124.

[3] Graefe's Archiv, 1882, Bd. xxviii., Abth. III., S. 201.

[4] Ueber die Gesichtsfeld und Faserverlauf im optischen Leistungs-Apparat, Halle, 1884.

[5] Archiv für Psychiatrie und Nervenkrankheiten, Bd. xiii. S. 352.

[6] Loc. cit.

[7] Graefe's Archiv, 1892, Bd. xxxviii., Abth. I., Ss. 1–70.

[8] The Toxic Amblyopias, Philadelphia, 1896, pp. 53–104.

[9] It is difficult to obtain statistical information on this subject. Some authors maintain that chewing tobacco causes amblyopia as frequently as smoking; others, for example, Ramsay, Charles W. Kollock, and Lautenbach, assert that they have never seen tobacco-amblyopia in a patient who chewed tobacco but did not smoke. This also has been the author's experience.

[10] Consult Berry, Transactions of the Ophthalmological Society of the United Kingdom, 1887, vol. vii. p. 72; and Nettleship, Royal London Ophthalmic Hospital Reports, 1886–87, vol. xi. p. 70.

[11] Journal of the American Medical Association, 1890, vol. xiv.

[12] International Medical Magazine, 1894–95, vol. iii. pp. 178–181.

[13] Consult also Dowling, Cincinnati Lancet-Clinic, 1892, vol. xxix. pp. 589, 590. A contrary conclusion was reached by E. T. Ely, New York Medical Journal, April, 1880.

vision, or, in other words, the maximum daily safe dose, varies from thirty grammes (Hirschberg) to fifteen grammes (Groenouw). In rare instances one-half ounce of strong tobacco per week has produced amblyopia.

An important factor is the *weight* of the tobacco, for example, of the cigars. Other things being equal, cigars heavy in weight are more likely to produce visual disturbance than those light in weight.

Smoking while the stomach is empty, continual smoking, illustrated by the expression "never without a pipe in his mouth," inhalation of the smoke, which thus comes in contact with a large surface for absorption, as, for instance, in cigarette-smoking, increase the dangers of a toxic influence, dangers which are manifestly heightened if the individual has characteristic susceptibility,—that is, an idiosyncrasy to the influence of the drug.

The *relation of nicotine* to the production of amblyopia has not been determined, except that it is believed that tobacco rich in nicotine—that is, tobacco of an inferior quality—is more apt to produce amblyopia than the higher grades, although the latter are not without danger. The great volatility of nicotine and the presence of numerous other poisonous substances in tobacco-smoke indicate, however, that nicotine cannot be the only active principle at work, and it is probable that the poisonous effects of tobacco, not only on the optic nerve, but on the system generally, are enforced by volatile alkaloids formed during its combustion. It seems not unlikely that *pyridine*, and less markedly *collodine*, should be regarded as active toxic agents in this respect.[1] The assertion that the paper in which cigarettes are wrapped is deleterious because it contains arsenic is not borne out by the analysis of M. Serog.[2]

It is quite possible that nicotine, or one or more of the many principles freely present in tobacco-smoke, liberate some toxic influence in the system which must be held accountable for the disease, which, in other words, depends upon a species of *auto-intoxication.* Horner long ago contended that neither tobacco nor alcohol, as such, was the direct toxic agent in cases of central amblyopia, but that together these drugs produced chronic gastric catarrh, which in its turn established a chronic anæmia of the optic nerve, terminating in the pathological changes which are found in this disease. Sachs[3] maintains that even in the pure tobacco cases certain complex chemical combinations occur in the stomach, and there is a resulting transformation of the normal gastric juices into acids of the fatty type, which combine with nicotine into substances which are more injurious than the simple tobacco bases themselves. This observation is important in connection with certain experimental work under the direction of Dr. Casey Wood, not yet published, which indicates that certain stomachic

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[1] For the further consideration of this subject, consult de Schweinitz, Toxic Amblyopias, p. 63; A. M. Ramsay, Lancet, May 11, 1895; Therapeutic Gazette, 1892, vol. xvi. p. 36.

[2] Quoted in the British Medical Journal, 1896, vol. ii. p. 1492.

[3] Archives of Ophthalmology, 1889, vol. ii. pp. 138–162.

toxines are capable of causing in animals blindness probably of the type now under consideration.

The *relation of alcohol* to the production of the amblyopia in the mixed cases seems to be this,—it predisposes to tobacco-amblyopia by producing chronic dyspepsia, or by lowering the vital powers, which are thus unable to resist toxic influences. It certainly is not antagonistic to the influence of tobacco, as was at one time taught by Jonathan Hutchinson. Amblyopia is also particularly liable to occur in those who are malnourished from other causes,—for example, chronic gastritis, mental worry, and particularly loss of sleep.

Certain races—for example, the Turks, and the Spaniards on the island of Cuba—appear to enjoy a comparative immunity from this affection and to withstand the evil influences of excessive inhalation of smoke. Negroes, however, according to Kollock, are not immune.

The *age* at which tobacco-amblyopia develops varies from the twenty-sixth to the seventy-fourth year. In rare instances it has been seen in patients under twenty, Berry reporting one case at the age of nineteen. Indeed, Lautenbach maintains that the affection is more common among young men than is generally supposed, one of his patients being a boy aged thirteen, who presented typical symptoms of tobacco-amblyopia.

As the disease is uncommon before the thirty-fifth year, the *time* required to produce toxic amblyopia consumes a number of years, generally fifteen or more. Exceptional instances of rapid development are on record. The author has observed one case after three years of smoking, and Nettleship reports one in a man who had smoked only for a year. F. C. Hotz, instead of a slow development of the amblyopia, has seen it appear with great suddenness.

The *relative frequency* of tobacco-amblyopia, owing to the difficulty of separating tobacco from alcohol cases, is hard to determine. Hirschberg states that tobacco-amblyopia comprises 0.6 per cent. of the whole number of toxic cases. Among twelve thousand six hundred and forty-four hospital patients examined by Priestley Smith, the percentage of tobacco-amblyopia was 1.20, while among fifteen hundred of his private patients it was 0.85. The percentage, according to the investigations of the author, in American hospital practice has varied from 0.10 to 0.25. In his private practice the percentage is about 0.47.

Symptoms.—Either with or without other symptoms of chronic tobacco-poisoning,—for example, restlessness, sleeplessness, disturbance of the circulatory equilibrium,[1] etc.,—the patient complains of diminution of sight, associated with fogginess in the centre of the field of vision, unimproved by glasses. The visual acuity is reduced from 20/30 to counting fingers

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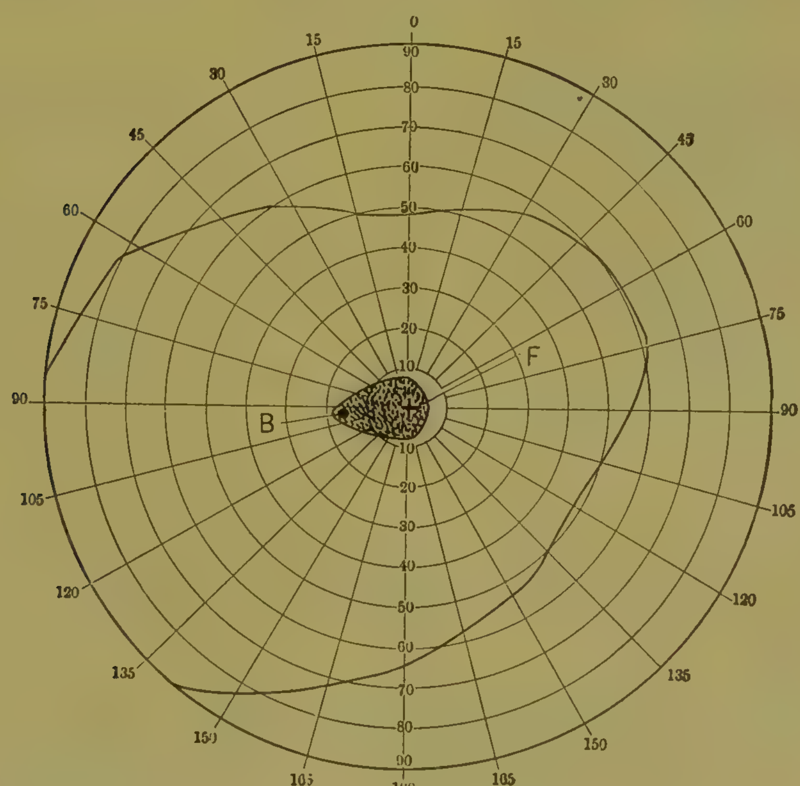
[1] In rare instances tobacco-amblyopia is associated with peripheral neuritis of the spinal nerves. Consult Judson S. Bury on "Peripheral Neuritis from Tobacco," Lancet, 1896, vol. ii. p. 28.

(according to Groenouw, from 20/30 to 5/200). Vision is better in dull light than in a bright glare, due, probably, to slight hyperæsthesia of the retina. Sometimes the near vision is particularly defective, while the far vision is reasonably good. Occasionally exactly the reverse is the case. There is nothing characteristic in the iris movements, and in very rare instances only do disturbances of the external ocular muscles occur. (Fontan.)

In the early stages the ophthalmoscopic findings are negative, or at most there is slight veiling of the edges of the disk and discoloration of its surface. Later there is pallor of the temporal half, or pallor of a quadrant-shaped area in the lower and outer part, an area which in aggravated cases assumes the appearances of complete atrophy.

Very exceptional ophthalmoscopic appearances are actual neuritis (Ponti), neuritis and retinal hemorrhage (Nettleship, A. S. Morton), and fine macular changes (Hill Griffith, Nettleship, Nuel, W. G. Laws, and the author). In some of these cases, however, there have been albumin and tube-casts, and the relation of the tobacco toxæmia to them has not been determined. These changes are of importance in view of the belief that the earliest lesions in tobacco-amblyopia consist in degeneration of the ganglion cells of the macula.

Fig. 5.

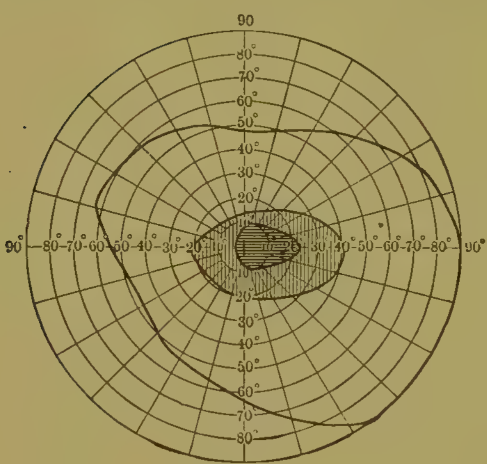



Average papillo-macular scotoma.—*F*, fixing-spot; *B*, physiologic blind spot.

Of greatest importance in the symptomatology are the visual field phenomena. They are as follows. The peripheral boundaries of the visual field are normal, and in the centre of each visual field there is a color

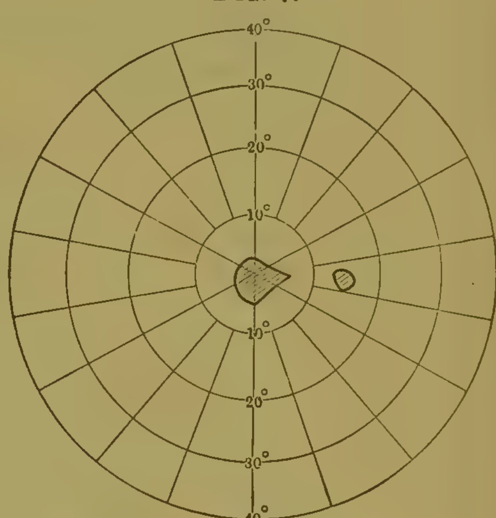
scotoma, especially for red and green, usually oval in shape, stretching from the fixing-point to the blind spot, and rarely passing much to the nasal side of the former. The average size of this *papillo-macular scotoma*, for so it is correctly described, according to Sachs's measurements, is outward eighteen degrees, inward five degrees, upward seven degrees, and downward six degrees. The author's average measurements are outward eighteen degrees, inward three degrees, upward seven degrees, and downward six degrees. (Fig. 5.) Therefore the scotoma is an oval with its pointed end towards the blind spot and its blunt end towards the fixing-point, to the nasal side of which it passes only slightly. This scotoma represents a red-green blind area, and commonly the extent of green blindness is greater than that of red, which in its turn may be surrounded by an area of imperfect color-sense. (Fig. 6.) The "culmination spot," or "nuclear spot," of the scotoma, to use the phraseology of Sachs, lies horizontally from one degree to eight degrees in a lateral direction from the fixation-point, its breadth vertically being mostly below the horizontal line. Sometimes, however, as Groenouw

FIG. 6.




Typical scotoma for red and green, represented by horizontal parallel lines, surrounded by an area of imperfect color-perception.

FIG. 7.




Small scotoma confined to fixation-point, often overlooked. (Groenouw.)

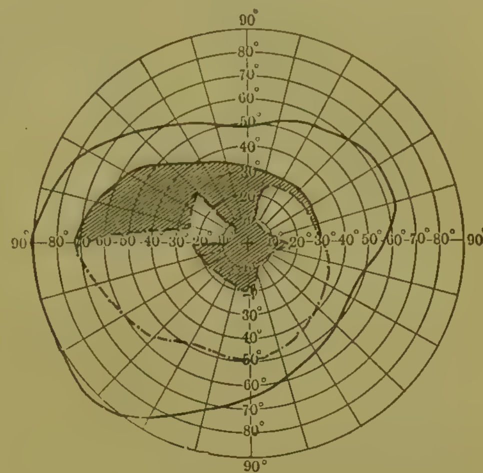
has pointed out, and as the author can substantiate, the beginning of the visual defect is a small, easily overlooked scotoma exactly over the fixing-point. (Fig. 7.)

The typical egg-shaped scotoma is formed, according to Groenouw, by a union of the scotoma from the fixing-point with a supplemental scotoma around the blind spot. Here the process may cease and restitution of vision occur with the disappearance of the color defect, retrogression gradually taking place from each end, the last area to regain perfect color-sense being that which has been occupied by the nuclear spot. On the other hand, there may be a stage of progression characterized by increase in the size of the color defect, usually above, until it meets the limit of the red

field,—that is, the scotoma has "broken through." (Fig. 8.) If this process goes on, the patient may eventually resemble a congenitally color-blind person.

In severe cases scotomas for blue and yellow form in similar manner to the red-green scotomas, especially, according to Baas,[1] in the period of "breaking through." The blue scotoma may be associated with xanthopsia. (Simi.) Finally, small absolute defects may be found particularly at the nuclear spot, but also elsewhere, and in neglected cases, or in those not typically toxic, the scotoma becomes absolute. (Fig. 9.) Bjerrum maintains that in all cases of intoxication-amblyopia within the color scotoma a smaller scotoma for white may be demonstrated if minute test-objects are employed.

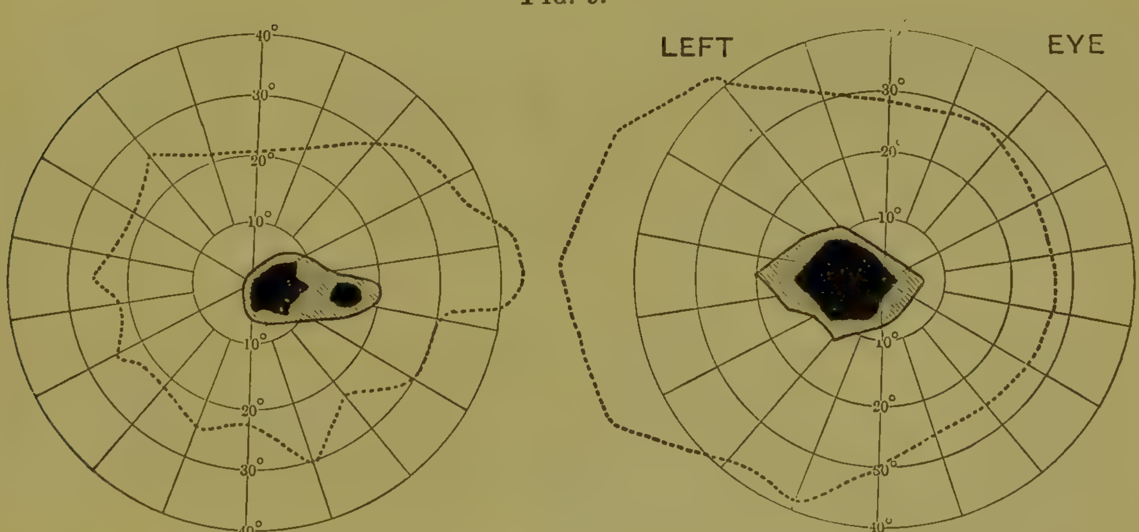
Fig. 8.



Breaking through of the scotoma.

Instead of the egg-shaped or oval scotoma, the visual defect may pass up and out, or down and out. Occasionally a circular scotoma surrounding the fixing-spot has been described,—for example, by Nettleship, Nelson, Doyne, and the author. Still more rarely, the scotoma may appear as a

Fig. 9.




Absolute defects within relative scotoma. (Groenouw.)

wedge-shaped color defect, somewhat dislocated from the fixing-point, beginning at about five degrees from its temporal side and passing to twenty degrees beyond this point, as in a case recorded by the author.

Although symmetry of the visual defect is the rule, some cases exhibit a certain degree of asymmetry, and a few so-called unilateral scotomas have been described. The author has seen two such cases, but, with Dr.

[1] Das Gesichtsfeld, Stuttgart, 1896, S. 164.

Berry, doubts whether they should be catalogued with the tobacco cases. George Ferdinands,[1] however, believes that in its earlier stages tobacco-amblyopia is as frequently asymmetrical as it is symmetrical, and adduces four cases in support of this view.

Intoxication-amblyopia without central scotoma has been described by Vossius. Berry has noted decided defects of the light-sense in abnormal states of the macular fibres, and suggests this method of examination as a diagnostic test of intoxication-amblyopia. W. Abney[2] has found marked diminution of the final sensitiveness to light of the central portion of the eye in cases of tobacco-amblyopia when compared with that of a person possessing normal light-sense, but R. Wallace Henry[3] concludes that cases of amblyopia with normal fundus, or, at most, slight pallor of the temporal side of the disk, should be classified as "toxic" if the light-perceptive power is normal, and that nicotine should be considered the determining cause if the visual field is full. If the light-perceptive power is four or under (5.24 being normal), the case is not regarded as a pure toxic amblyopia.

**Diagnosis.**—This depends upon the history of the case, the visual field phenomena, and the exclusion of locomotor ataxia, diabetes, and insular sclerosis, particularly the last two diseases, and the effects of certain poisons, prominent among which are alcohol, bisulphide of carbon, iodoform, and stramonium. The development, enlargement, and retrogression of the toxic scotoma are often analogous to the same phenomena in the scotoma of non-toxic retrobulbar axial neuritis, only that in the latter the defect is absolute and often positive, except in the beginning, and tends to pass to the nasal side of fixation and does not assume specially a horizontally oval shape. The scotoma which occurs with locomotor ataxia is similar to the toxic variety, but progressive, as are also the optic nerve changes, and the same may be said of that variety which occurs with scotomatous atrophy of the disk. Much more difficult is the differentiation between the toxic scotomas and those caused by disseminated or insular sclerosis, and the author does not doubt that the latter affection, which in its early stages may sometimes manifest itself only by visual phenomena, has been mistaken for the former. The scotomas are very similar, and the differentiation must be made by the history and by general examinations.[4] An examination of the urine will reveal the diabetic cases.

**Prognosis.**—The prognosis in pure tobacco-amblyopia, or in the mixed varieties in which the poisons have not been used for any great length of time, is always good, provided the patient will stop the abuse of tobacco, because, as Nettleship has pointed out, perfect recovery does not

[1] British Medical Journal, 1896, vol. ii. p. 653.

[2] Proceedings of the Royal Society of London, 1891, vol. xlix. p. 49.

[3] Ophthalmic Review, 1896, vol. xv. pp. 33–43.

[4] On this point consult Mr. Marcus Gunn, Transactions of the Ophthalmological Society of the United Kingdom, 1897, vol. xvii. p. 120.

ensue without at least a diminution of the ordinary allowance of tobacco.[1]

Relapses in tobacco-amblyopia, according to Groenouw, Nettleship, Eales, and other observers, are uncommon, some authors stating that they never occur,—that is, that the patient may smoke again after cure with impunity. While the author has observed this apparent immunity from a second attack, he has also seen well-marked relapses, and Meyer[2] maintains that relapses are frequent if the patient is again exposed to the original injurious conditions, and that the case may terminate in progressive atrophy.

There seems no doubt that in a certain number of instances optic nerve atrophy has resulted from tobacco-amblyopia, although, as in the alcoholic cases, complete blindness, if it occurs at all, must be rare. Again, there is a type of optic nerve atrophy in which tobacco, while not the sole cause of the affection, certainly has some share in determining it. Such cases have been particularly described by Edgar A. Browne, J. B. Lawford, and the author. If vision is reduced below one-tenth of normal and the scotoma begins to encroach upon the nasal side of fixation, the chances for complete recovery, as Dr. Berry has stated, are poor. The surgeon, however, should not despair of reaching a good result because improvement is delayed, since we know from the observations of A. H. Thompson[3] that improvement may be very slow and cure result many months after the appearance of the original affection.

### (c) *Alcohol-Tobacco Amblyopia.*

Only exceptionally the opportunity comes to separate tobacco from alcohol cases, inasmuch as most smokers are also drinkers of spirits. Therefore the majority of the cases of this type of toxic amblyopia belong to the *mixed variety*, the influence of one or other drug usually predominating, the consensus of opinion at present being that tobacco is more potent than alcohol in its deleterious effects upon vision. Necessarily the symptoms of the mixed variety would be a repetition of those already recited.

**Pathology and Pathological Anatomy of Toxic (Central) Amblyopias.**—We are not in possession of the results of a post-mortem examination of a perfectly pure case of intoxication-amblyopia, because all of the patients thus far examined suffered from various types of wide-spread disease.[4] The author's endeavors to establish toxic (alcoholic) amaurosis in

[1] A few authors have denied the influence of tobacco in producing these scotomas; for example, Minor in this country and Filehne in Germany. The mass of evidence is so overwhelmingly against their views that it is unnecessary to discuss them in detail.

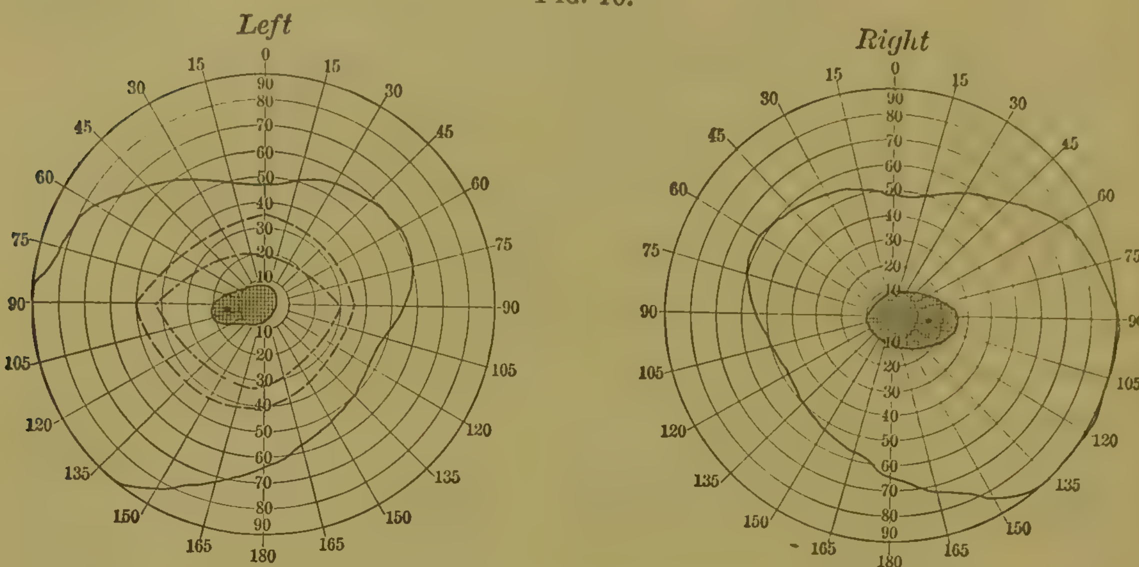
[2] Traité pratique des maladies des yeux, Paris, 1895, p. 283.

[3] Royal London Ophthalmic Hospital Reports, 1897, vol. xiv., Part II.

[4] Widmark (Mittheilungen aus der Augenklinik des Carolinischen medico-chirurgischen Instituts zu Stockholm, Jena, 1898, Ss. 1–19) has recorded a case of alcohol-tobacco amblyopia in a man aged fifty-four, who committed suicide, which may be regarded as an exception to this statement. There was complete degeneration of the papillo-macular bundle. Unfortunately, there is no description of the post-mortem condition of the internal organs.

monkeys and dogs were failures, probably because the drug was not continued for a sufficiently long period.

Fig. 10.




Visual fields of the patient from whose optic nerves the sections illustrated on Plates I. and II. were obtained.—The continuous line indicates the limits of the form fields; the broken lines the color fields, — — — blue; —·—·— red; the central cross-hatching the scotomas. There was green blindness. It was difficult to ascertain the limits of the color fields in the right eye; they are, therefore, not represented. The exact limits of the scotoma were also difficult to determine; they probably reached the boundary of the red field above.

The test-objects were white and colored circles, one centimetre in diameter. The scotomas were determined with one-quarter centimetre square colored tests.

In the *early stages* of tobacco-amblyopia marked improvement, as A. M. Ramsay points out, often follows a good night's rest, while visual deterioration is caused by fatigue. Such symptoms are not consistent with the theory that any portion of the optic nerve is seriously inflamed or actually degenerated at this period of the disease. They may be explained by assuming that a *vascular disturbance* or a *transient œdema* in the form of an exudation from the blood-vessels has appeared in the macular fibres, which, being functionally the most active, are the first to be affected by pressure or other conditions which have been suggested. That the early stages of this affection are in some degree dependent upon vascular disturbance seems further indicated by the influence of nitrite of amyl in causing improvement in vision. Indeed, Silcock and Broadbent have actually observed a scotoma disappear in diabetic amblyopia (which depends upon analogous lesions), when under the influence of this drug a previously high arterial tension was lowered to normal. If the process is not arrested at this time, what may be described as a functional derangement in the macular fibres passes over into an organic change, resulting finally in tract atrophy of the character presently to be described.

Another explanation is suggested by the theory—now receiving histological proof—that the earliest lesions in intoxication-amblyopia are to be found in the ganglion cells of the retina and in the nerve-fibre layer. It is readily conceivable that the macular ganglion cells may suffer from a lesion sufficient to cause visual disturbance, which disappears, with consequent im-

provement in acuity of sight, under the influence of rest, improved hygiene, etc., precisely as we know that the constitution of ganglion cells elsewhere in the body is demonstrably structurally different after enforced exercise and electrical stimulation from what it is after rest. If improper stimulation is long continued, permanent lesions are established in these cells. So also if the influence of the toxic agent is exerted for undue periods of time, degeneration of the macular ganglion cells ensues together with secondary changes in the optic nerve.

Based upon the anatomical proof of more than twenty autopsies, associated with careful microscopical examinations, the situation of the pathological process in central amblyopia in its *later stages* is determined to be in the optic nerve, and especially in that portion of it which is known as the *papillo-macular bundle* or *macular fascicle*.[1]

This process apparently may start at different points. Both Sachs and Samelsohn note that the origin is in the optic canal, the former observer believing that the intensity of the atrophic changes shades off both towards the brain and the globe. In two of Uhthoff's cases the changes in the nerve reached the optic foramen and entered the orbital cavity; in others they were present only in the distal end of the orbital portion of the nerve at its entrance into the eye and from six to twelve millimetres posterior to this point. In the specimens examined by the author (see Plates I., II.) it is evident that the entire papillo-macular bundle is degenerated, and that it pursues a course similar to the one already described by Samelsohn, Vossius, Uhthoff, and other observers. The lesion appears in a wedge-shaped area of degeneration on the temporal side of the optic nerve, which position it maintains, although in a somewhat altered shape, being at first heart-shaped and later crescentic for about ten millimetres behind the globe. The area then approaches, but never quite gains, the centre of the nerve, and maintains this position until it reaches the foramen. In the intercranial portion of the nerve the patch again becomes distinctly crescentic and occupies a position above the centre. In the chiasm the foci of degeneration are symmetrically placed slightly below the centre, while in the tract the position is almost exactly central. The degeneration both upon the right and the left side is more marked in two situations,—namely, just posterior to the lamina cribrosa and again at the optic foramen,—although just before reaching the latter point there is an area with patches of well-preserved nerves.

This preservation of normal nerve-fibres has been noted by Uhthoff in the midst of the atrophic region in *alcoholic neuritis*, but is not found in simple gray atrophy of the nerve when it affects only a portion of the structure. Other differences between simple gray atrophy, as it occurs in tabes and in progressive paralysis, and the lesion which is found in central amblyopia, according to Uhthoff, are to be found in the appearances, at least

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[1] See this System, vol. ii. p. 240.

## DESCRIPTION OF PLATES I. AND II.

### Plate I.

1.—*Longitudinal Section of the Posterior Half of the Right Bulbus and Five Millimetres of the Optic Nerve.* The degenerated area occupies strictly the temporal half of the nerve and passes to the pial sheath. Close to the lamina the degeneration is complete; farther onward there are traces of normal nerve-fibres. (Fig. 1, Plate I.)

2.—*Transverse Section of the Right Nerve Eight Millimetres behind the Globe.* The degenerated patch occupies the temporal (lower and outer) portion of the nerve forming a somewhat heart-shaped area, extending at one point to the sheath. This area is imperfectly divided by a faint line of partially preserved nerve-fibres. (Fig. 2, Plate I.)

3.—*Transverse Section of the Nerve Thirteen Millimetres behind the Globe.* The degenerated patch, still occupying the lower and outer part of the nerve, becomes more contracted and assumes an oval or somewhat meniscus-like shape. (Fig. 3, Plate I.)

4 and 5.—*Transverse Sections of the Nerve in the Region of the Optic Foramen.* The degenerated patch tends to leave the periphery and pass to the centre, although still somewhat eccentrically placed. (Fig. 4, Plate I.) Farther onward it is still more centrally placed, with the broader end towards the outer border. (Fig. 5, Plate I.)

6.—*Transverse Section of the Nerve in the Intra-Cranial Region.* The degenerated patch is somewhat crescentic and with the broader end towards the temporal side. It occupies a position somewhat above the centre of the nerve. (Fig. 6, Plate I.)

PLATE I.

Fig. 1.

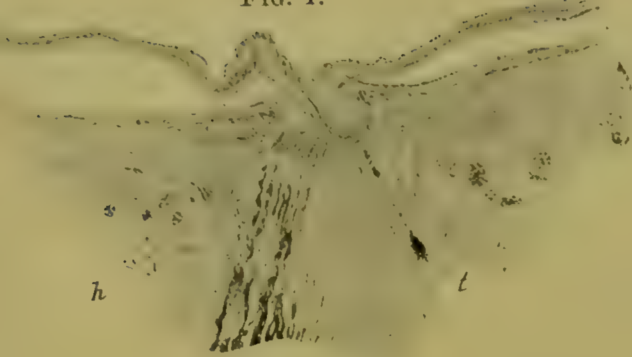



Fig. 2.

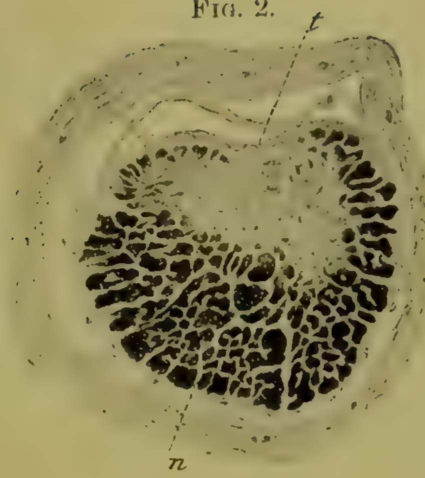



Fig. 3.

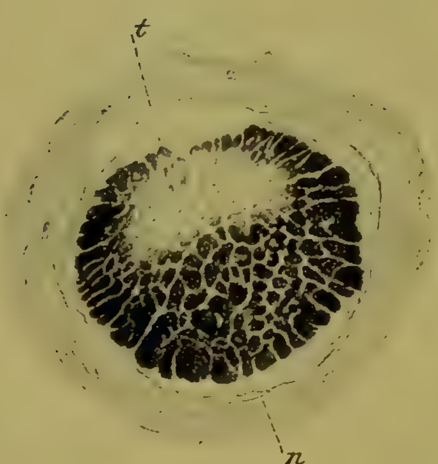



Fig. 4.

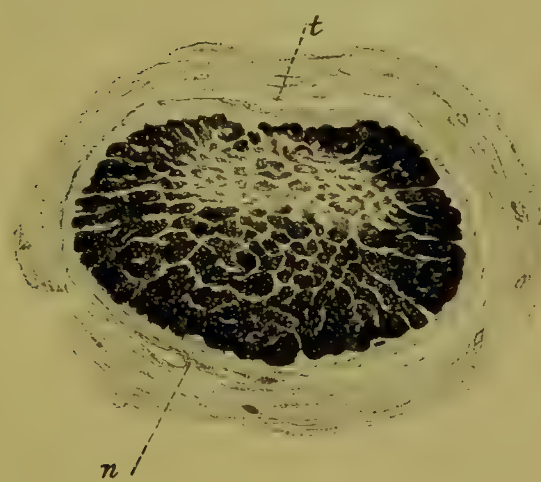



Fig. 5.

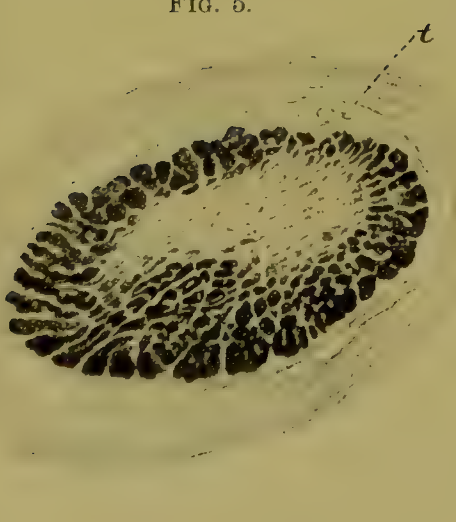



Fig. 6.

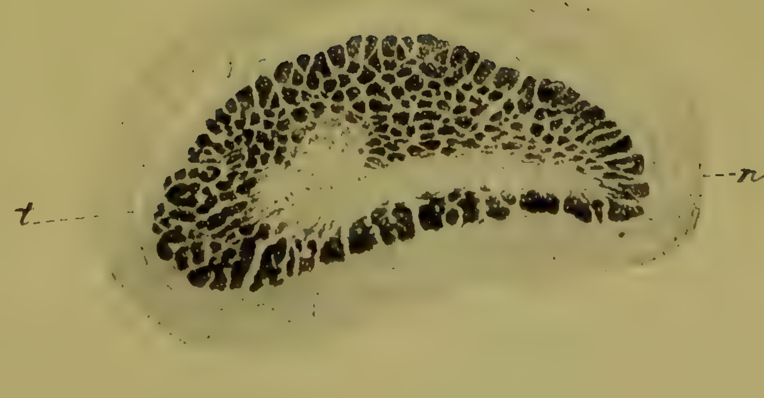


PLATE II.

Fig. 1.

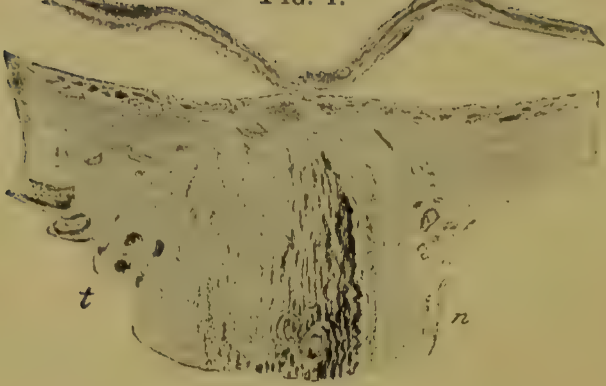



Fig. 2.

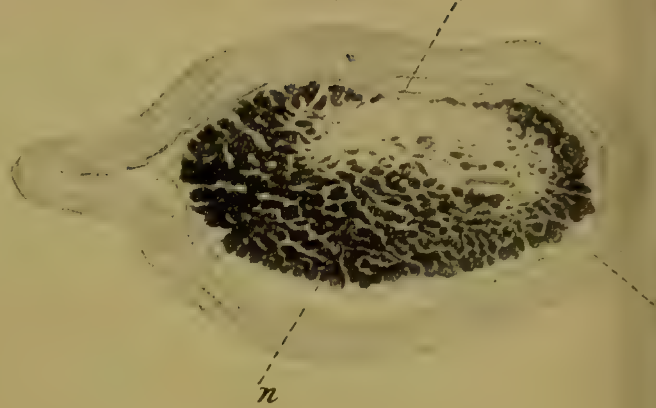



Fig. 3.

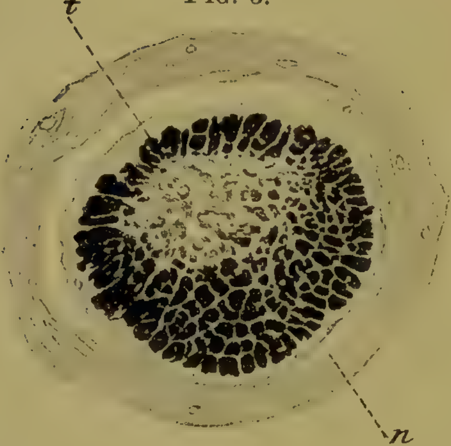



Fig. 4.

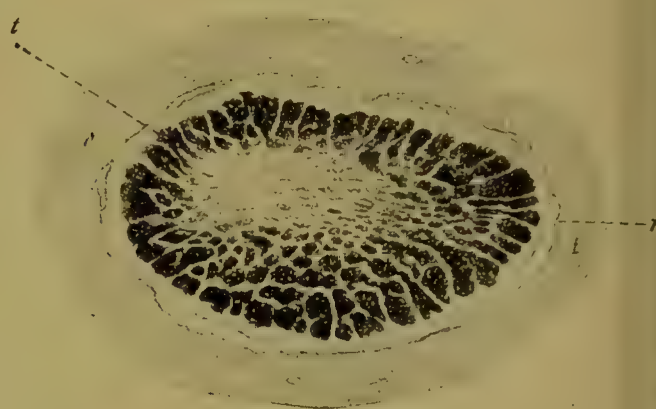



Fig. 5.

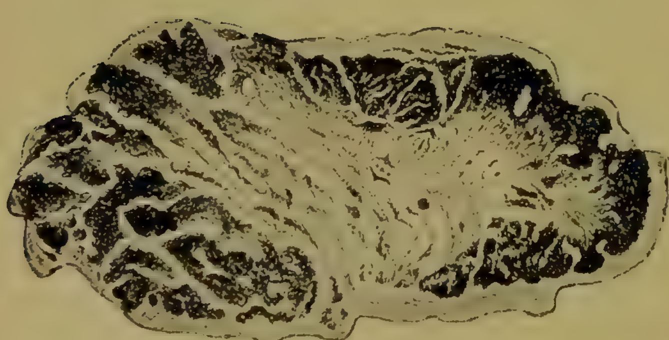


Fig. 7.

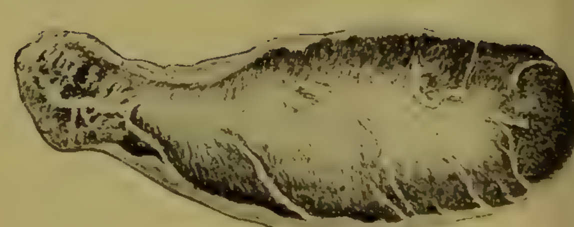
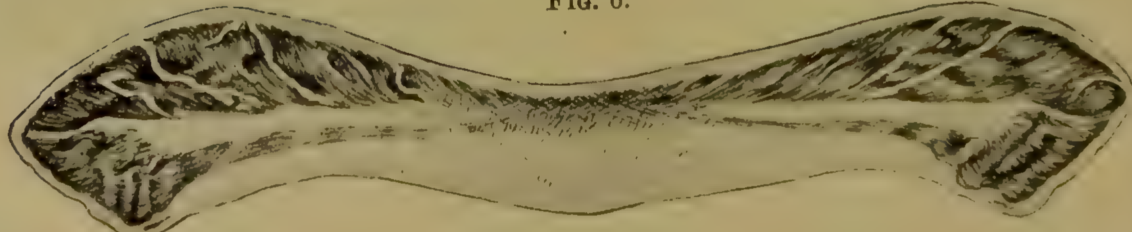


Fig. 6.



## Plate II.

1.—*Longitudinal Section of the Posterior Half of the Left Bulbus and Eight Millimetres of the Optic Nerve.* The degenerated area again strictly occupies the temporal half of the nerve and passes to the pial sheath. Close to the lamina the degeneration is almost complete. Farther towards the brain patches of retained nerve-tissue are seen. If the patch is studied in its relation to the vessels, it is seen in certain sections to occupy a somewhat triangular patch, which, in cross-section at this point, would produce the well-known wedge-shaped patch with the apex towards the vessels which has so often been described. (Fig. 1, Plate II.)

2.—*Transverse Section of the Left Nerve Ten Millimetres behind the Globe.* The degenerated patch forms a crescentic or somewhat meniscus-like area with its concavity towards the vessels and reaching at its outer border quite to the pial sheath. It is divided by a distinct line of retained normal nerve-fibres, the line running in the long axis of the patch. (Fig. 2, Plate II. Compare with Fig. 3, Plate I.)

3 and 4.—*Transverse Sections of the Nerve in the Neighborhood of the Optic Foramen.* The degenerated patch again tends to leave the periphery and reach the centre (Fig. 3, Plate II., compare with Fig. 4, Plate I.), which it more nearly attains at the foramen (Fig. 4, Plate II., compare with Fig. 5, Plate I.), its broadest end being towards the temporal side. The remainder of the nerve to the chiasm tallies closely with that upon the right side, as does also the appearance of the optic tract. Therefore, figures and descriptions need not be reproduced.

5.—*Transverse Section of the Right Nerve Just in Advance of the Chiasm.* The section is somewhat broken, but shows the degenerated patch occupying an irregular area in the centre of the section, and reaching below to its margin. (Fig. 5, Plate II.)

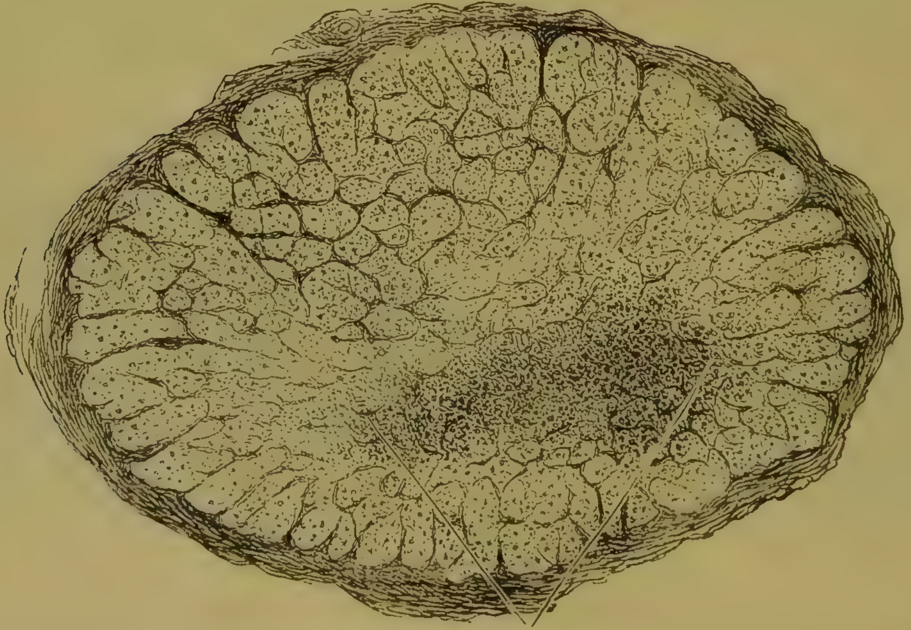
6 and 7.—*Transverse Sections of the Chiasm and Right Optic Tract.* The degenerated patches occupy symmetrical positions in either end of the ellipse, being nearly centrally placed and gradually narrowing to a point of decussation in the centre of the chiasm. (Fig. 6, Plate II.) In the optic tract the degenerated area at first occupies almost exactly the centre of the tissue, and becomes less and less marked as the connection between this tissue and the midbrain is being formed. (Fig. 7, Plate II.)

in the earlier stages, of the connective tissue of the nerve in its finest ramifications, which in gray atrophy is not thickened, but in alcoholic neuritis shows thickening and proliferation.

Samelsohn, Vossius, Uhthoff, and others emphasize the inflammatory character of the toxic amblyopia-process, at least so far as its operation in the optic canal is concerned, but Sachs believes that the preponderance of connective tissue which he found is not due to an interstitial inflammatory hyperplasia, but is a secondary formation caused by the collapse and falling together of the connective-tissue septa after the loss of the nerve tissue between them.

The vascular changes, in general terms, may be said to consist of thickening of the small nutrient vessels of the optic nerve, similar changes in the major vessels, and, according to Sachs, endo- and periphlebitis of the vena postica and choking of peripheral capillaries and small extravasations in their neighborhood. The marked thickening of the interfascicular septa, the vascular lesions, and the increase in nuclei, both upon the edge of the degenerated area and within the fasciculi, even if this increase in nuclei cannot be said always to be a marked one, present appearances which closely correspond with those usually assigned to an interstitial sclerosing inflammation, which Samelsohn compared with the same pathologic process visible in interstitial hepatitis. (Fig. 10 *b*.) As has been well stated, however, "the descriptions of the optic nerves in the recorded cases agree quite

FIG. 10 *a*.

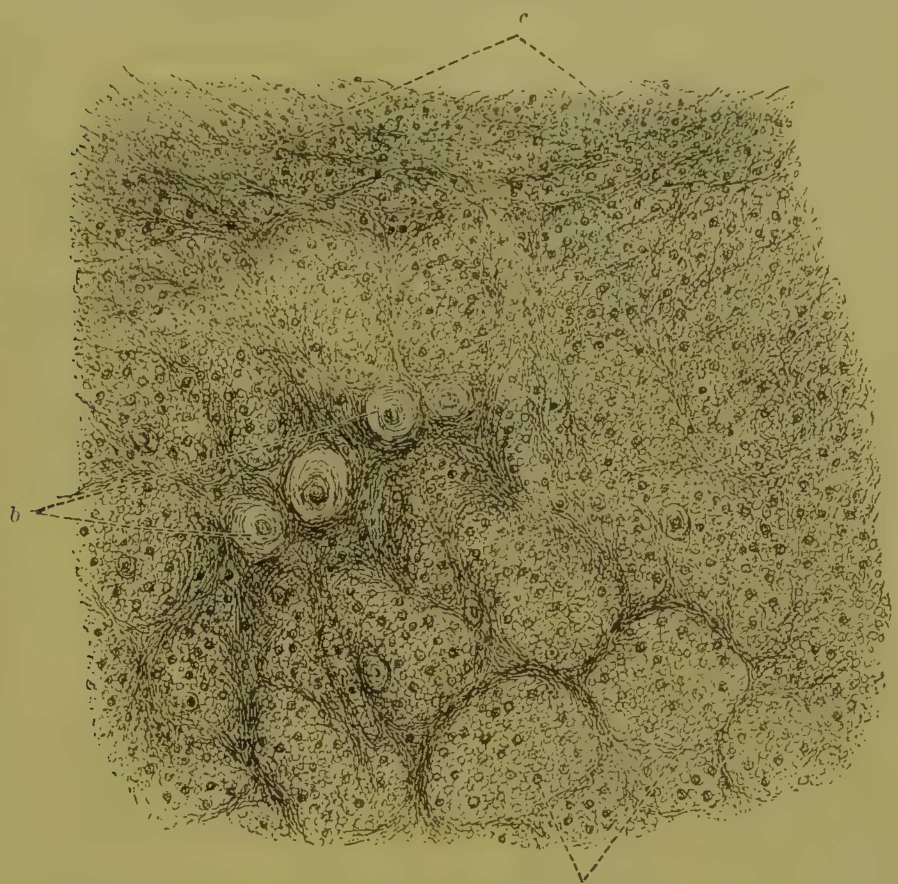



Transverse section of the optic nerve in toxic retrobulbar neuritis, 15 mm. behind the globe, showing at *a* oval patch of degeneration and small-celled infiltration. $\times$ 90. From a case of tobacco-amblyopia.

as well with an inflammation of the connective tissue of the nerve, causing secondary atrophy of the nerve-fibres, as with a primary atrophy of the latter associated with secondary interstitial changes following in its track."

Not all authors have been satisfied with the explanations of the pathogenesis of toxic central scotoma just given. Schoen[1] strongly advocated the retinal origin of central amblyopia, and believed that the scotoma was

Fig. 10 b.




Portion of diseased area in toxic retrobulbar neuritis. × 240.—a, nerve-bundles with beginning infiltration of brightly staining nuclei; b, markedly thickened arteries; c, thickening of interfascicular septa and collapse of nerve-bundles.

the functional expression of a physiological weakness of the centre of the retina heightened by chronic intoxication. Similar views have been expressed by Treitel and Baer. Recently Nuel[2] has contended that the central toxic scotoma is not primarily the result of a neuritis of the macular bundles, but of a disease of the macula lutea, causing degeneration of its cells, and that the optic nerve changes are secondary to destruction of the nerve-cells of the macula. He bases his views on microscopical examinations of a specimen in which the ganglion cells were atrophied, and recent investigations, referred to on page 835, strongly indicate that degeneration of these cells is to be expected in toxic amblyopia. There is no doubt that there may be a retinal origin of degeneration of the papillo-macular bundle, or, indeed, of more extensive disease of the entire nerve, and clinically we know that in atrophic central retino-chorioiditis, or in so-called macular coloboma, there will be a quadrant atrophy of the optic nerve, precisely as this

[1] Die Lehre von Gesichtsfeld, Berlin, 1874, S. 116.

[2] Archives d'Ophtalmologie, October, 1895; March and August, 1896.

is seen in cases of intoxication-amblyopia. Moreover, Usher and Dean[1] have produced macular fibre degeneration in a monkey, exactly as it occurs in intoxication-amblyopia, by making a lesion in the retina between the yellow spot and the optic disk. Ophthalmoscopically changes in the macula have been described, although they were not attributed to the influence of the toxic agent. (See page 805.) Sachs and the author have carefully examined the ganglion cells of the macula in cases of toxic amblyopia and found them normal, or at least only slightly changed, although the entire macular tract was atrophied; but their investigations were made with the ordinary histological methods and before the Nissl stain was known. Hence their value is indifferent. In Widmark's case (*loc. cit.*) the nerve-fibre layer between the macula and papilla was thinner than normal and the ganglion cells somewhat less numerous.

The central or cortical situation of the affection has been advocated by Filehne,[2] himself the subject of tobacco-amblyopia, but without good grounds on which to base this belief.[3]

**Treatment of Intoxication-Amblyopia.**—If the disease has not advanced too far, absolute cessation from smoking and drinking, particularly from the former habit, will be sufficient to effect a cure. In other cases remedies which have the power of favorably altering the vascular, inflammatory, and degenerative processes must be used. Of these the most important are mercury, iodide of potassium, and strychnine. Mercury may be employed in the form of the bichloride, or preferably by inunctions. Temporary improvement follows the administration of nitrite of amyl. In the later stages, or if the ophthalmoscopic appearances of atrophy have begun, nitro-glycerin and strychnine should be pushed to their physiological limits. Marked improvement will follow free sweating, either by the administration of the hydrochlorate of pilocarpine, from one-tenth to one-quarter of a grain hypodermically, or by inducing diaphoresis by mechanical methods,—the Turkish bath, etc. Gastric irritation, dyspepsia, and insomnia must be combated by appropriate remedies.

The length of time required for a cure varies, according to the extent of the poisoning and the faithfulness of the patient in following directions, from a few weeks to a number of months. If actual atrophy ensues, the treatment of this affection is the same as that advised for atrophy of the optic nerve from other causes, and, in the judgment of the author, should

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[1] Transactions of the Ophthalmological Society of the United Kingdom, 1896, vol. xvi. p. 275.

[2] Graefe's Archiv, 1895, Bd. xxxi. ii. S. 1.

[3] A curious theory with reference to the pathogenesis of tobacco-amblyopia has been advanced by R. W. Doyne (Royal London Ophthalmic Hospital Reports, 1888–89, vol. xii. p. 51). This author thinks that tobacco and other agents may have a toxic influence on the visual purple, degenerating it and causing retinal exhaustion, which shows itself in the failure of the more delicate color-sense. The exhaustion naturally takes place at the point of greatest retinal activity and where the light is proportionately stronger, the rays being more accurately focussed.

include the administration of strychnine, nitro-glycerin, and bichloride of mercury. Electricity, in his experience, is of little value, although some authorities place great reliance upon the employment of voltaic alternatives. Subconjunctival injections of corrosive sublimate have also been used. With them in this affection the author has no experience. Recently De Wecker has advocated the treatment of toxic amblyopia by serum therapy.

### RELATION OF TOBACCO AND ALCOHOL TO ACQUIRED COLOR-BLINDNESS.

As already described, when there is progression of the central toxic scotoma and it breaks through, meeting the limit of the red field above, the patient's condition resembles that of a congenitally color-blind person; but even in the earlier stages of this affection the scotoma might interfere with the proper recognition of signal lamps by railroad employees and sailors. Thus, Priestley Smith states that tobacco-amblyopia fully developed might prevent a man from recognizing the color of a distant lamp, although he still might properly differentiate the colors of the skeins of wool used in testing the color-sense. Therefore, in all examinations of railroad employees, any defective vision not readily improved by optical therapeutics should be investigated by the careful search for scotomas with small colored test-objects.

### TOBACCO AMBLYOPIA AMONG THE LOWER ANIMALS.

Although some animals—for example, the Virginia deer—eat the leaves of the tobacco plant without detriment, having become immune to its influence by reason of many years' residence in the tobacco district, there is some evidence to show that other animals (horses) suffer from a form of amblyopia due to the consumption of tobacco. For our knowledge of this subject we are indebted chiefly to Dr. J. W. Barrett, of Melbourne, Australia.[1] In certain regions of New South Wales horses are attacked by a form of amaurosis which has been attributed to their consumption of some plant, probably the Australian tobacco (*Nicotiana suaveolens*). As, however, some of the horses show also paresis or paralysis of the hind legs, it is probable that the blindness is part of a wide-spread affection of the nerves. Dr. Barrett's investigations show that the optic nerves are degenerated and wasted. Through his courtesy the author was enabled to examine a microscopical section of the optic nerve of a horse blinded, it was supposed, by the consumption of this tobacco plant. The slide was composed of about one hundred bundles, of which at least eight showed distinct signs of disease,—namely, a species of fibrosis which separated, pressed upon, and destroyed the individual fibres. This was a marked phenomenon in several of the bundles and less apparent in others. This observation appeared to confirm in large measure those already made by Dr. Barrett, who also found atrophy of the nerve-fibres, but who does not describe

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[1] The Australasian, July 7, 1894; and Intercolonial Medical Journal of Australasia, 1897, vol. ii. No. 4.

over-development of connective tissue. It appeared exactly to coincide with the observations of Dr. Tidswell, who describes the condition as one "of progressing fibrosis with some degeneration of the nerve-fibres." A similar blindness has been ascribed to the influence of the grass lily and the native melon.

## AMBLYOPIA FROM ESSENCE OF JAMAICA GINGER AND FROM ESSENCE OF PEPPERMINT.

The first case of this affection was reported by Dr. A. G. Thomson.[1] The essence of Jamaica ginger is used to excess by sailors, oystermen, and others in place of whiskey, and after a debauch lasting from two to five days, during which great quantities of the drug are consumed,—according to the statement of one patient, as much as twelve ounces in twenty-four hours,—acute blindness supervenes, lasting for several days, followed by a restoration of vision, to be again succeeded by central amblyopia and progressive atrophy of the optic nerve. Through the courtesy of Dr. William Osler, of Johns Hopkins University, the author has investigated one such case, finding beginning contraction of the peripheral field, absolute central scotomas of irregular shape and much larger than those found in pure toxic cases, and the ophthalmoscopic appearances of progressive atrophy of the optic nerve without preceding neuritis.

It is stated, on lay authority only, however, that precisely similar conditions may arise from the excessive use of *essence of peppermint*, several cases of this character having been reported in the northwest of the United States among Indians who have consumed large quantities of the drug in place of alcohol.

## AMBLYOPIA FROM BISULPHIDE OF CARBON AND CHLORIDE OF SULPHUR.

**History.**—The poisonous effects of bisulphide of carbon on the workers in that branch of the india-rubber manufacture in which the rubber is vulcanized by immersion in a solution of chloride of sulphur in bisulphide of carbon was first systematically described by Delpech in 1856,[2] and since this date many additional cases have been recorded in France, Belgium, Germany, and England. In the last-named country a special committee was appointed by the Ophthalmological Society of the United Kingdom[3] to specially investigate this subject. So far as the author is aware, only one case has been carefully examined in this country, and has not been reported.[4]

**Etiology.**—Toxic symptoms are probably always the result of the

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[1] The Ophthalmic Record, 1897, vol. vi., n. s., pp. 579–582. Dr. Hiram Woods informs the author that he has examined several cases.

[2] Mémoire sur les accidents que développe dans les ouvriers en caoutchouc l'inhalation du sulfure de carbone en vapeur, Paris, 1856.

[3] Transactions of the Ophthalmological Society of the United Kingdom, vol. v. p. 157.

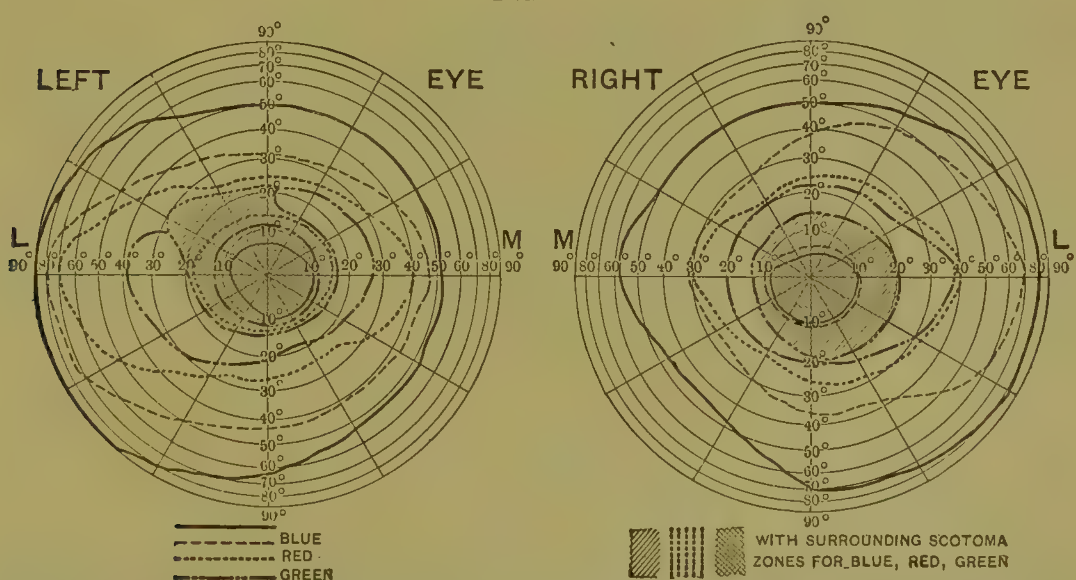
[4] Personal communication by Dr. Henry Ring, of New Haven.

workman's exposure to the fumes of the fluid used in the caoutchouc manufacture, and of the two agents, chloride of sulphur and bisulphide of carbon, the latter is the more noxious. Amblyopia occurs in about forty per cent. of the cases of chronic bisulphide of carbon poisoning, which has been noted from the fifteenth to the sixty-sixth year. Males are more frequently exposed to the vapors, and therefore are more frequently affected than females.

**Symptoms.**—Somewhat characteristic of this amblyopia, as S. Reiner,[1] who has made the most recent analysis of the subject, points out, is that the amblyopia is practically always preceded by the general nervous symptoms of the poisoning.[2] The ocular symptoms are a mist before the eyes, followed by decided lowering of central visual acuity (counting fingers, or seeing only large type, J. 16 or 20), dilated pupils, impaired power of accommodation, and ophthalmoscopically general pallor of the optic disk, pallor of the temporal half (Reiner), perineuritis, slight neuritis, or complete atrophy. A normal fundus has been described by Gallemaerts. Hirschberg has found stippling in the macular region analogous to that seen in eyes of patients poisoned with naphtalin.

At first the field of vision is normal, or nearly normal, in its peripheral boundaries, and there is a central scotoma, which is most marked for red and

FIG. 11.




Amblyopia from bisulphide of carbon poisoning.—Absolute central defect, surrounded by a scotoma-zone for blue, red, and green. (Uhthoff.)

green, but may also be present for white, blue, and yellow. The scotoma may be minute and central (Fuchs), absolute, central, and surrounded by a scotoma-zone for blue, red, and green (Uhthoff), (Fig. 11), ring-shaped,

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[1] Wiener klinische Wochenschrift, 1898, Bd. viii. S. 919.

[2] Those interested in the general phenomena of bisulphide of carbon poisoning should consult Delpech (loc. cit.), Fuchs (Transactions of the Ophthalmological Society of the United Kingdom, 1884–85, vol. v. p. 152), and Ross (Medical Chronicle, Manchester and London, 1886–87, vol. v. p. 257).

or paracentral, or massive and extending especially to the temporal side of the median line (Becker). Occasionally, instead of the scotoma, there is red-green blindness, and sometimes only contraction of the color-fields. One author reports normal color-perception (Dumont).

**Diagnosis.**—This is ascertained by the history of the case. According to the committee of the Ophthalmological Society of the United Kingdom, although slight toxic symptoms may be noticed within a few days, the date of the onset of severe symptoms, including the failure of vision, is very variable.

**Prognosis.**—This depends upon removal of the patient from his poisonous surroundings. In the author's collection of cases fifteen recovered or greatly improved, five were unimproved, and the result was not stated in three. In the collection of the Ophthalmological Society thirty-three per cent. recovered, twenty-five per cent. improved, and twenty per cent. showed little or no improvement.

**Pathological Anatomy.**—Autopsies are lacking, and, therefore, it may be assumed from the clinical symptoms that the lesion is a retrobulbar or axial neuritis, analogous to that found in tobacco-amblyopia. It is interesting, however, to notice Hirschberg's observation of macular changes, and future investigations may show that the optic nerve lesions are secondary to disease of this area.

**Treatment.**—The chief treatment is prophylactic, and the workmen in caoutchouc manufactories should be provided with an apparatus to prevent inhalation of the fumes, while every opportunity to secure perfect ventilation in the curing-rooms of such manufactories should be taken advantage of. Special forms of apparatus, or respirators, have been devised. The workmen should be advised against the use of tobacco and alcohol, because Becker's observations indicate that these substances predispose them to the amblyopia. The medicinal treatment of the affection is the same as that already detailed in connection with tobacco-alcohol amblyopia.

## AMBLYOPIA FROM IODOFORM.

**History.**—The first case of amblyopia attributed to iodoform was recorded by Hirschberg,[1] and since this date additional cases have been reported by E. Hutchinson, of Utica, New York,[2] Priestley Smith,[3] Valude,[4] Terson,[5] James W. Russell,[6] and Anderson Critchett.[7] The ages of the cases reported were respectively twelve, sixteen, twenty-six, thirty-one, thirty-two, and forty-eight. In two cases (Hutchinson's and Critch-

[1] Centralblatt für praktische Augenheilkunde, 1882, Bd. vi. S. 92.

[2] New York Medical Journal, 1886, vol. xliii. p. 16.

[3] The Ophthalmic Record, 1893, vol. xii. p. 101.

[4] Annales d'Oculistique, 1893, t. cix. p. 378.

[5] Archives d'Ophtalmologie, October, 1897.

[6] Lancet, 1897, vol. i. p. 1608.

[7] Transactions of the Ophthalmological Society of the United Kingdom, 1898.

ett's) the age is not given. The sexes are equally divided,—four females and four males. The influence of tobacco and alcohol was practically excluded in each case. Four of the patients (those of Hutchinson, Smith, and Russell) were treated with iodoform pills for pulmonary complaints (phthisis), while in the others the iodoform was used in one instance (Hirschberg's) as a dressing for a diseased hip, in two others for extensive burns, and in one for a cancerous breast. The quantity of the drug necessary to produce amblyopia appears to be considerable, Smith's patient consuming one thousand grains in forty-one days. On the other hand, Russell's patients exhibited amblyopia after ten-grain doses three times a day for three weeks.

**Symptoms.**—The visual acuity has varied with the severity of the case from 6/18 to counting fingers, and the ophthalmoscopic appearances in the milder cases have been practically normal, or perhaps only slight pallor of the disk. In others the nerve-head may resemble that seen in tobacco-alcohol amblyopia, or there may be slight haziness of the disk margin, or, finally, complete atrophy. A normal field of vision and the absence of color scotomas may be expected in milder forms of this affection,—for example, such as have been recorded by Hutchinson and James W. Russell. In more advanced cases there is a bilateral central color scotoma, which may be absolute near the fixing-point, and intact peripheral visual field, while in the cases with complete optic nerve atrophy there is color-blindness. Critchett reports a scotoma for blue.

**Diagnosis and Prognosis.**—The diagnosis necessarily must be based upon the history of the case. The prognosis, if complete atrophy has not set in, is good, the duration of the amblyopia varying from eight days to several months. In two of the cases—namely, those reported by Valude and Terson—there were extensive burns, and therefore it is doubtful exactly what influence the drug itself had in the production of the amblyopia, because it is well known that neuro-retinal lesions may follow wide-spread cutaneous burns. In both of these instances, however, the histories indicate that the iodoform and not the burn was the potent agent in producing the amblyopia.[1]

**Pathological Anatomy.**—Judging from the clinical symptoms, the affection seems to depend upon some influence of the drug directly upon the fibres of the optic nerve, and particularly upon the papillo-macular bundle, or else upon the ganglion cells of the retina. Michel, commenting on Smith's case, seems to regard it as one of tubercular disease of the nerve-head, without, apparently, however, the least evidence on which to found this assertion.

**Treatment.**—This consists in removal of the patient from the influence of the drug, and the internal administration of strychnine, nitrite of amyl,

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[1] For a full discussion of the relation of burns to this lesion the reader should consult the papers of Terson and Valude.

and the other remedies which have already been mentioned in connection with the management of cases of toxic amblyopia. The strychnine treatment appears to have been promptly successful in two of the instances recorded.

### AMBLYOPIA FROM IODURET AND THIURET.

Two cases of this affection have been reported by Karl Baas.[1] The first patient, a boy of fifteen, after an operation for osteomyelitis of the femur, was treated locally with the antiseptic substance known as *ioduret*. At the end of three weeks there were marked diminution of central visual acuity, normal boundaries of the white field, contraction of the blue field, and a central scotoma. Later the papilla assumed a greenish-white color, particularly upon the temporal side. The second case was a sixteen-year-old girl whose surgical lesion was a wide-spread burn, which was treated by applications of *thiuret*, and a month later she had marked failure of central vision, a large central scotoma, and temporal atrophy of the optic nerves. Removal of the drug and the injection of strychnine resulted in improvement. The analogy of this case to those in which iodoform was used in the treatment of burns and resulted in central amblyopia is striking.

### AMBLYOPIA FROM NITROBENZOL AND DINITROBENZOL.

**History.**—Nitrobenzol used in the manufacture of aniline, soaps, etc., and dinitrobenzol employed in making explosives, have the power, in addition to creating violent general symptoms of poisoning, of bringing about a form of toxic amblyopia to which attention was first drawn by Nieden.[2] Simeon Snell[3] has contributed a thorough paper on the ocular lesions of dinitrobenzol amblyopia, together with the record of five cases.

**Etiology.**—The poison may enter the system through the mouth, through the lungs, by inhalation of the fumes or the fine dust, or through the skin, and, according to Snell, the presence of impure products increases the danger of working with nitrobenzol, the most injurious work being that of grinding and mixing. Age, sex, and previous general condition appear to exercise no special influence on the liability to the poisonous effects of these substances. Sometimes the symptoms of poisoning appear rapidly, sometimes gradually after some months of occupation in the manufactories.

**Symptoms.**—In addition to the general symptoms of nitrobenzol-poisoning, those specially related to the eye are diminished central acuity of vision, contraction of the color-field, reversal of the color-lines (Nieden), occasionally a central color scotoma for red and green (Snell), and ophthalmoscopic changes which consist in a dark color of the fundus, moderately overfilled arteries, venous hyperæmia, pale or blurred optic disks, and small

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[1] Das Gesichtsfeld, Stuttgart, 1896, Ss. 173–177.

[2] Centralblatt für praktische Augenheilkunde, 1888, Bd. xii. S. 194.

[3] British Medical Journal, 1894, vol. ii. p. 499.

retinal hemorrhages. The venous hyperæmia and discoloration of the fundus may be present in those who work in manufactories employing dinitrobenzol, even though they have no visual disturbance.

**Diagnosis.**—The amblyopia somewhat resembles that produced by bisulphide of carbon, but the peculiar appearances of the fundus are the distinguishing characteristics and are not present in any other form of toxic amblyopia.

**Prognosis.**—If the patient can be removed from his work the prognosis is good, although the restoration to perfect vision may consume some months. The serious general disturbances which this poison produces are of more moment in the prognosis than the visual defects.

**Pathological Anatomy.**—The pathology of these cases is not settled.[1] So far as the eye is concerned there is evidently a vaso-motor paralysis, as Nieden suggests, which accounts for the overfilling of the veins. The symmetrical character of the visual defects, together with the color scotomas, indicates axial disturbance in the optic nerve. The author has suggested that as this and similar toxic agents have the property of changing the blood to a deep chocolate brown and causing it to lose its power of absorbing oxygen, their influence may be credited in some way to these blood-changes, very much as we believe that the blood in certain dyscrasias possesses toxic or infective qualities capable of acting upon retinal vessels previously disposed towards alterations in their walls and lining membrane.

**Treatment.**—The patient must be removed from the noxious influence of the poison, and the case managed on the general principles which have now several times been enunciated. The preventive treatment is of the greatest importance, and Simeon Snell's rules, that the different processes in the manufactories should be conducted as much as possible in the open air, that in "mixing" closed vessels should be employed, that some form of respirator should always be used, and that the chemicals should not be touched with bare hands, should be strictly followed.

## VISUAL DISTURBANCES FROM CHRONIC LEAD-POISONING.

**History.**—Disturbances of vision due to the toxic influence of lead have probably been known for nearly three hundred years, one of the earliest accounts having been published by Henricus Smetius in 1611. The "mysterious colic of the ancients," associated with amaurosis, has been attributed by Tanquerel and Grisolle to lead. After the introduction of the ophthalmoscope the alterations in the nerve-head and fundus oculi generally began to be noted, particularly by Hirschler,[2] Hutchinson,[3] and Breurer,[4]

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[1] Those interested in the general effect of these chemicals upon the system should consult Ross (Medical Chronicle, 1889, vol. x. p. 89), and White (The Practitioner, 1889, vol. xliii. p. 14).

[2] Wiener medicinische Wochenschrift, 1866, Bd. xvi. Ss. 105–121.

[3] Royal London Ophthalmic Hospital Reports, vols. vi. and vii.

[4] Ueber Amblyopia saturnina, Bonn, 1876.

who reviews the cases up to the date of the publication of his thesis. In more recent times thorough communications on this subject, with reviews of the literature, both ancient and modern, will be found in the papers of Stood,[1] Von Schroeder,[2] the author,[3] and Elschnig.[4]

Etiology.—The lead may enter the system either with articles of food or drink contaminated by being stored in leaden vessels, or through the skin by handling paints or other substances composed of white lead, or by using hair-dyes, cosmetics, etc. Occasionally the source of contamination is most obscure; for example, lead-poisoning may occur in seamstresses who bite instead of break the threads they use in sewing when these threads have been weighted with sugar of lead, and instances have been reported from chewing shot;[5] but by far the greatest contingent of those affected comes from house-painters and workers in white lead manufactories or in chromate of lead.

Far more males than females have been affected, probably because more males than females are employed in lead-works. Of ninety cases gathered by the author, sixty-five were males and twenty-two females. In three the sex is not mentioned. The age is not stated in eighteen, and ranges from twelve to seventy-nine years in the remaining seventy-two. The length of time which may elapse before lead causes toxic symptoms varies considerably, from a few weeks to many years; but even when other general symptoms of saturnine toxæmia are present, amblyopia is, comparatively speaking, rare. Thus, among forty-eight lead-workers examined by Dr. Frederick Packard,[6] the average length of time of exposure among those working in the lead for more than a year being nine and a half years, twenty-four had at one time or another distinct symptoms of lead-poisoning, but no case of amblyopia directly traceable to the metal could be discovered.

Lead-amblyopia, when compared with the whole number of cases of ocular disease examined, is uncommon. Among nearly fifteen thousand case records in the author's hospital practice during the last five years, only three undoubted cases have presented themselves. As compared with the number of cases of toxic amblyopia from other causes, that produced by lead represents a small percentage, only one case of lead-amblyopia being found among one hundred and thirty-eight cases of toxic amblyopia analyzed by Uhthoff.

Symptoms.—Usually preceded by other signs of chronic plumbism, the ocular symptoms of the lead toxæmia may manifest themselves as,—

1. Transient amblyopia, without ophthalmoscopic change, resembling uræmic amaurosis.

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[1] Graefe's Archiv, 1884, Bd. xxx., Abth. III., S. 215.

[2] Ibidem, xxxi., Part I., pp. 229–249.

[3] Toxic Amblyopias, 1896, pp. 149–163.

[4] Die Ophthalmologische Klinik, May 5, 1898.

[5] Charlotte (N. C.) Medical Journal, 1896, vol. viii. pp. 178–180.

[6] Philadelphia Hospital Reports, 1896, vol. iii. p. 38.

2. Amblyopia without fundus lesions, or with congestion of the nerve-head, and with central scotomas analogous to those occurring under the influence of other toxic agents.

3. Optic neuritis, or neuro-retinitis, either specifically due to lead or secondary to changes in the brain or kidneys.

4. Optic nerve atrophy, either consecutive to a plumbic papillitis or retrobulbar neuritis, or due to a primary effect of the lead on the visual organ.

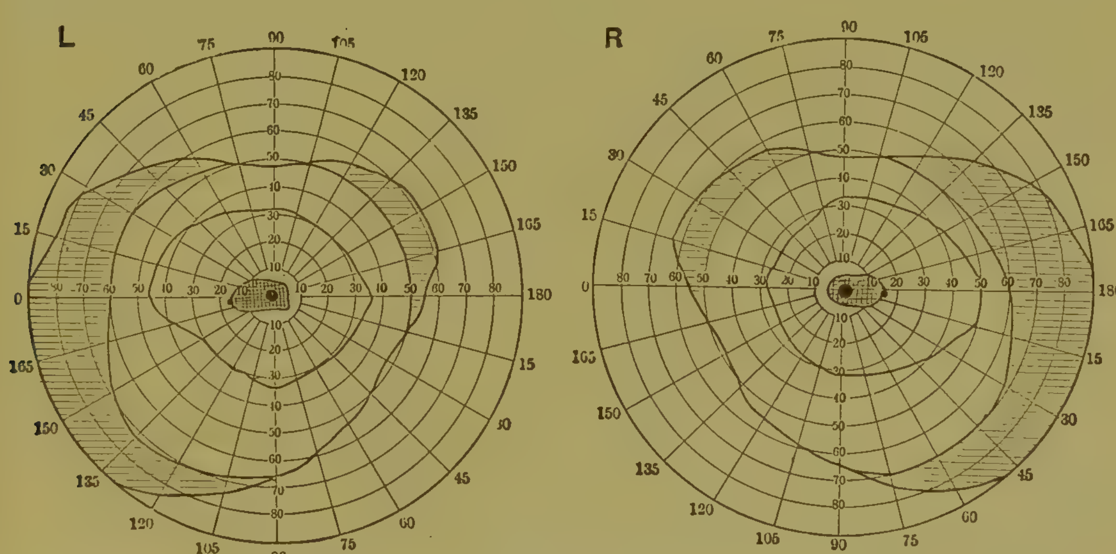
5. Various types of retinitis, vasculitis, and perivasculitis, either primarily due to lead or secondary to nephritis.

Judging from the investigations of Stood and the author, in fully fifty per cent. of the cases of lead-amblyopia organic changes may be expected in the optic nerve.

Visual acuity varies, according to the character of the fundus changes, from practically normal sight to absence of light-perception. The iris movements are governed by the condition of the optic nerve apparatus; paralysis of the external ocular muscles is not uncommon.[1]

The field of vision may present the following alterations: concentric contraction for form and colors, contraction and peripheral relative scotomas (Stood), and typical central scotomas, such as are found in other cases of intoxication-amblyopia (Uhthoff, the author). (Fig. 12.) Optic neuritis with bitemporal hemianopsia has been reported. (Westphal, Elschnig.)

FIG. 12.




Central scotomas from lead-amblyopia with peripheral contraction of the visual field.—Small absolute defect within the relative area.

**Diagnosis.**—There are no ocular symptoms diagnostic of chronic plumbism, and the various lesions which have been described can be attributed to lead only when this toxic agent has been proved to be present by chemical examinations, or when other sources of poisoning have been eliminated.

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[1] Consult an excellent paper by Casey A. Wood, the Medical News, 1897, May 29; also Elschnig, loco citato.

**Prognosis.**—Necessarily this depends upon the character of the lesion, the duration of the process, and the accuracy of therapeusis. In an analysis of seventy cases of all varieties of ocular disturbance due to lead, there was no improvement in twenty-seven; recovery is stated to have occurred in thirty-three.

**Pathology and Pathological Anatomy.**—Certain authors—for example, Jeaffreson[1]—deny the specific action of lead upon the visual apparatus, and assert that the various types of neuritis and retinitis should be ascribed to renal disorders, effusion into the brain and subarachnoid spaces, and in women to menstrual derangements caused by the lead. While many cases of such character occur, there is no doubt, as Thomas Oliver has well shown, that visual disturbances of the kind described are also specifically due to lead.

In general terms, the ocular lesions in chronic plumbism are peripheral in character, and the retrobulbar neuritis of lead-poisoning, as Stood expresses it, is peripheral and analogous to the condition which the metal causes in the musculo-spiral nerve (wrist-drop). The central scotoma indicates that in some cases the papillo-macular bundle is early and especially affected, although the process may not stop here, but go on until the intermediate and peripheral fibres are involved and general optic nerve atrophy results. According to Parisotti,[2] the nutrient arteries of the optic nerve and the vessels of the retina suffer from a form of endovasculitis which he terms *endarteritis saturnina obliterans.* This explains the various types of retinitis and those cases of optic nerve atrophy without preceding neuritis, as well as the fibrous, hyaline, and fatty metamorphosis of the tissues supplied by these vessels. Transient lead amaurosis may be caused by an anæsthetic effect of the poison on the retina, or may be due, according to Elschnig, to a spastic ischæmia of the retina.

**Treatment.**—The free administration of iodide of potassium is the most useful therapeutic measure. Baths of sulphuret of potassium have some repute. Should optic nerve atrophy supervene, the usual remedies for this condition are indicated. The preventive treatment is that in vogue in all well-regulated lead-works,—free ventilation, the use of respirators and gloves, and frequent washing of the hands and mouth, the former with water slightly impregnated with sulphuric acid.

## AMBLYOPIA FROM ARSENIC-POISONING.

It is not a little remarkable, when the wide-spread pathological lesions which occur in chronic arsenical poisoning are taken into consideration, that so few well-authenticated cases of optic nerve and retinal disturbance appear to be on record. The drug may reach the system through the skin,

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[1] British Medical Journal, 1886, vol. i. p. 390.

[2] Recueil d'Ophtalmologie, 1885, 3 s., t. vii. p. 350. Consult also Oeller, Virchow's Archiv, 1881, Bd. lxxxvi. S. 329.

the stomach, or by inhalation through the lungs, and has produced amblyopia with the following lesions: retrobulbar neuritis with sector-discoloration in the temporal half of the optic papilla and paracentral negative scotomas for red and green (Liebrecht);[1] optic neuritis (H. Derby);[2] optic nerve atrophy (Feilchenfeld).[3] So far as the author is aware, cases of amblyopia among the arsenic-eaters of Styria are not reported. Workers in arsenic, paper-hangers, etc., in addition to other visual disturbances, suffer from conjunctival hyperæmia and eczema of the lid, and the prolonged use of the drug is said by Hutchinson, Sen., to cause vitreous opacities.

## AMBLYOPIA FROM NITRATE OF SILVER.

According to Gowers, amblyopia, analogous to that caused by lead, may be one of the symptoms of *argyria*, and it is stated by Reimer that silver has been found in the sclerotic sheath of the optic nerve. But cases of toxic amblyopia in the true sense of the word do not seem to be reported with accuracy, Bresgen's case being distinctly untrustworthy and poorly observed.

## AMBLYOPIA FROM MERCURY.

By the older writers—for example, Haffner, Himly, and Deval—mercury is always credited with the power of producing defective vision. Temporary amblyopia has been reported by Dyes[4] after a fourteen days' inunction with gray salve. Optic neuritis has been ascribed to the influence of mercury by Square, and optic nerve atrophy by Galezowski; but, with Leber, we doubt whether the etiological relationship between these affections and the mercury has been properly established.

MacAdam[5] describes amaurosis from *mercuric methyde*, which contains eighty per cent. of the vapor of mercury, but gives no particulars. In *acute mercury-poisoning* it is probable that hemorrhages and fatty degeneration of the retina will occur.

**Treatment.**—This should include the internal administration of iodide of potassium, and afterwards measures which are suited to the management of optic nerve atrophy. The same treatment applies to arsenic-amblyopia.

## AMBLYOPIA FROM PHOSPHORUS.

This occurs because early in its toxæmia there are retinal hemorrhages and later fatty degeneration of the retinal tissue itself, which, should the patient live long, would no doubt produce secondary changes in the optic nerve. The influence of acute poisons upon the retina and the secondary changes in the optic nerve require further investigation.

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[1] Klinische Monatsblätter für Augenheilkunde, 1891, Bd. xxix. S. 181. In this case the influence of tobacco, alcohol, and syphilis was not excluded. The supposed source of poisoning was the consumption for a long period of time of arsenical pills.

[2] Boston Medical and Surgical Journal, 1891, vol. i. 124, p. 603.

[3] Beitrag zur Kasuistik der Atrophia nervi optici. Inaugural Dissertation, Kiel, 1896.

[4] Zeitschrift für praktische Heilkunde, 1865, Bd. ii. S. 260.

[5] Edinburgh Medical Journal, 1866, vol. xii., Part II., p. 718.

## AMBLYOPIA FROM VARIOUS COAL-TAR PRODUCTS.

Intense pigmentation of the cornea and conjunctiva (aniline kerato-conjunctivitis), with reduction of vision, is not uncommon among workers in ANILINE dyes, and has recently been especially studied by Senn.[1]

A deficiency in visual acuity, reduction of the amplitude of accommodation, hemeralopia, and even iritis have been reported by Galezowski, Manouvrier, Kohn, and others among workmen who are required to handle and prepare the various coal-tar products. The amblyopia may be associated with discoloration of the papilla and reduction of the size of the retinal vessels,[2] or with slight optic neuritis and negative central scotomas. (C. A. Veasey.[3])

Of the various products, probably aniline is the most active in producing poisoning, either alone or as an impurity in nitrobenzol. FUCHSIN has also been credited with similar toxic properties, but probably inaccurately. SAFRANIN is decidedly poisonous, and may be responsible for some of the visual disturbances.

According to Bitter, CREOLIN may cause "dull vision." NAPHTALIN in rabbits will produce opacities in the vitreous, fatty degeneration of the retina, and secondary cataract; but similar lesions are not produced in human beings, nor does the drug give rise to a typical toxic amblyopia.

ANTIFEBRIN (acetanilid) in large dose (forty-five grains), according to Hilbert,[4] may cause complete blindness, followed by marked anæmia of the optic nerve and retina, with shrinking of the vessels and contraction of the visual field. The similarity of this condition to quinine amaurosis (page 833) becomes at once apparent, and would suggest a similar pathogenesis. Other symptoms noted after large doses of this drug have been narrow and immobile pupils (Simpson) and distention of the retinal veins (Müller).

ANTIPYRIN (phenozone), according to Goodman, may produce temporary amaurosis. The author has observed visual disturbances characterized by undulations in the atmosphere, phosphenes, and diminished accommodative power. No doubt phenomena of this character, as well as the temporary amaurosis, should be ascribed to disturbance in the blood-supply of the retina.

## AMBLYOPIA FROM OPIUM OR ITS ALKALOIDS.

The older writers—for example, Beer and Himly—give opium a prominent place among those drugs to the influence of which they were wont to attribute amaurosis. It appears that the excessive use of opium, or large doses of this drug consumed within comparatively short periods of time,

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[1] Abstract in the American Journal of the Medical Sciences, 1897, N. S. 114, p. 115.

[2] Consult Galezowski, Des amblyopies et des amauroses toxiques, Paris, 1878.

[3] In Veasey's case the dye-worker was exposed to several sources of poisoning, with aniline as the most active agent.

[4] Therapeutische Wochenschrift, 1897, Bd. iv. S. 728.

may produce amblyopia, or even complete blindness, with slight veiling of the edges of the papilla and contracted and motionless pupils (W. Wagner[1]); or amblyopia with intact visual fields, due to irregular and spasmodic contraction of the muscle of accommodation (Galezowski); or temporary loss of sight, with contracted pupils, attributable to cramp of the retinal vessels (Hammerle[2]). Furthermore, according to Schiess-Gemuseus, there may be pallor of the temporal half of the disk, with right-sided half-blindness and left-sided concentric contraction of the visual field; or, as reported by Reymond,[3] atrophy of the optic papilla and central scotoma; or, as recorded by Grippo,[4] diminished central acuity and restricted visual field, with central scotomas. The last three cases indicate that opium may be classified with those drugs, clinically at least, which produce an intoxication-amblyopia with the lesion in the papillo-macular bundle.

## AMBLYOPIA FROM CHLORAL.

Visual disturbances have been ascribed to the influence of chloral, as follows: temporary amaurosis (Fischer-Dietschy and K. Murphy); amblyopia, faint color scotoma, and hyperæmic disk, which disappeared after the habit was stopped (Mittendorf[5]); intoxication-amblyopia with color scotomas and intact peripheral field (Levinstein[6]); and defective sight analogous to that seen in hysteria, due to paresis of the ciliary muscle (Förster). It must be admitted that the cases are indefinite and the etiological relationship of chloral to the amblyopia somewhat uncertain.

## AMBLYOPIA FROM THE POTASSIUM SALTS.

Among other symptoms of the long-continued use of *bromide of potassium* (bromism), keratitis (Gifford), phlyctenular conjunctivitis (Knies), and ptosis and dilated pupils (Rudisch) have been described; but intoxication-amblyopia, if it occurs at all, must be rare, apparently only one case of blindness, white nerve-head, and contracted arteries caused by excessive doses of the drug being on record (Reubel[7]).

With the exception of vague references in early writings, amblyopia attributable to *iodide of potassium* does not seem to have been reported. Solutions of *iodine* injected into the circulation at the base of the brain are said to cause amaurosis and narrowing of the retinal vessels. (Von Basch.)

*Chlorate of potassium*, it has been suggested, may cause amaurosis sec-

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[1] Klinische Monatsblätter für Augenheilkunde, 1872, Bd. x. S. 335.

[2] Deutsche medicinische Wochenschrift, 1888, Bd. xiv. S. 838.

[3] Abstract Annales d'Oculistique, 1873, t. lxix. p. 165. In this case chronic poisoning was not due to opium alone, but also to chloroform.

[4] La Riforma medica, 1895, iv., No. 2, p. 122.

[5] Medical Record, New York, 1889, vol. xxxvi. p. 134.

[6] Abstract Nagel's Jahresbericht, 1883, Bd. xiv. S. 302. In addition to chloral, morphine and nicotine appear to have been used.

[7] Partial blindness from the administration of potassium bromide and fluid extract of passion flower has been reported by Dr. Harnsberger.

ondary to the nephritis which is part of the pathology of its acute toxæmia.

Cases of amblyopia due to *cyanide of potassium* are not on record, except in so far as disturbances of the pupillary reflexes are concerned; but exposure to the vapor of dilute *hydrocyanic acid* has caused temporary amaurosis and hemiopia, due, no doubt, to disturbance either of the retinal circulation or of the cortical centres. (H. De Tatham.)

## AMBLYOPIA FROM CANNABIS INDICA.

In *acute* cannabis indica poisoning visual hallucinations, violet vision, dim vision, and depreciation of the amplitude of accommodation may occur.

In *chronic* cannabis indica poisoning (hashish habit) typical intoxication-amblyopia has been described by Ali,[1] characterized by central scotomas (sometimes monolateral) and intact peripheral visual fields.

## AMBLYOPIA FROM COFFEE, TEA, AND CHOCOLATE.

Certain cases of amaurosis were ascribed by the older writers to the abuse of coffee. In more recent times Jonathan Hutchinson is quoted as having seen a "coffee-amblyopia," without particulars.[2] Red vision and metamorphopsia in a child caused by drinking strong coffee have been reported by L. Connor. Excessive coffee-drinkers, in countries where this beverage is used in enormous quantities,—for example, in Arabia,—are said to become blind not infrequently from a form of optic nerve atrophy perhaps analogous to that caused by alcohol.

Tea-tasters may suffer from visual disturbances, and Dr. Berry,[3] in the list of substances which he believes may produce intoxication-amblyopia, includes tea as a possibility. Amblyopia with partial central scotomas for red, attributed to drinking huge quantities of strong tea, and improving after the beverage was stopped, has been recorded by E. Kenneth Campbell.[4]

Some of the earlier writers include *chocolate* among the substances capable of producing amaurosis, and Casey Wood[5] records a case of temporary amblyopia from chocolate, appearing in the form of a migraine, associated with a scintillating scotoma. The same writer suggests that minor defects of vision manifesting themselves in the form of asthenopia probably not infrequently are caused by drugs and beverages in every-day use. He includes in his list not only quinine, the salicylates, iodides, bromides, alcohol, etc., but tea, coffee, chocolate, high game, and strong cheese, believing

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[1] Recueil d'Ophtalmologie, 1876, p. 258.

[2] The author has been unable to verify this statement, and believes, although it often appears in literature, that it is a mistake, and that what Hutchinson really said is that he had seen deafness produced by the abuse of coffee.

[3] Transactions of the Ophthalmological Society of the United Kingdom, 1887, vol. vii. p. 91.

[4] The Lancet, 1898, vol. i. p. 717.

[5] Medical Record, New York, 1895, vol. xlviii p. 843.

that the asthenopic symptoms are occasioned by some form of dyspepsia which these substances originate; in fact, a form of ptomaine-poisoning.

### AMBLYOPIA FROM VANILLA.

According to Guérin,[1] the ocular disturbances from the ingestion of vanilla, or from handling the vanilla-pods, may appear in various forms of conjunctivitis, increased eyeball tension, "progressive diminution of vision which may go on to amaurosis," congested optic disks, and occasionally retinitis and chorio-retinitis. He compares the condition to a subacute glaucoma. Cure is effected by change of occupation, antiphlogistics, alteratives, and myotics, preferably pilocarpine.

### AMBLYOPIA FROM STRAMONIUM.

A true toxic amblyopia from affection of the papillo-macular bundle may be caused by smoking stramonium-leaves in great quantities, and reveal itself with the usual manifestation of blurred vision, colored scotoma, etc. Well-observed cases of this character have been recorded by Fuchs[2] and Cerrillo.[3] In the latter case the central scotomas were absolute and there was neuritis.

### AMBLYOPIA FROM ERGOT.

Amblyopia from ergot, in the sense of an intoxication visual disturbance, if it occurs at all, must be extremely rare. The author is acquainted with one case observed by Dr. H. V. Würdemann, of Milwaukee, with relative central scotomas, but alcohol, lithæmia, and tobacco could not be entirely excluded. During ergotism cataract is said to form,—the so-called raphanic cataract.

### AMBLYOPIA FROM CARBONIC OXIDE AND CARBONIC ACID.

Interstitial neuritis of the optic nerves, with inferior hemianopsia, has been observed by Raffegeau in carbonic oxide poisoning. It is probable that in this case there was a lesion in the occipital lobes. Schmitz, however, under similar circumstances, has described contraction of the visual field, partial color-blindness, venous hyperæmia in the retina, and contracted arteries.

### AMBLYOPIA FROM OSMIC ACID.

Amblyopia from osmic acid, owing to an action of the vapor of this drug upon the retina, has been reported by Dr. Noyes,[4] and one or two other examples of osmic acid amblyopia are upon record. Certain persons are very sensitive to its vapor, and after exposure vision becomes dim for

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[1] Annales d'Oculistique, 1895, t. cxiv. p. 292, English edition.

[2] Text-Book of Ophthalmology, American edition, New York, 1892, p. 441.

[3] Recueil d'Ophtalmologie, July, 1895.

[4] Transactions of the American Ophthalmological Society, 1866.

several hours, very much as if a mydriatic had been instilled in the eye. The same effect sometimes follows exposure to the vapor of *formalin.*

## AMBLYOPIA FROM PREPARATIONS OF THE CINCHONA BARK (QUININE-AMAUROSIS).

**History.**—The earliest records of visual disturbances caused by quinine are by Berandi,[1] Robach,[2] Beydler,[3] and Giacomini,[4] and in American literature by H. C. Lewis,[5] W. O. Baldwin,[6] and others. The first cases examined ophthalmoscopically are those reported by von Graefe,[7] but the earliest carefully recorded examples of the affection begin in 1879 with Voorhies's[8] and Roosa's[9] cases, which were soon followed by other reports, especially those by Knapp, De Wecker, Gruening, Michel, Buller, Dickinson, and many others, until now the literature comprises fully one hundred cases coming from almost all portions of the civilized world.

Long ago (in 1847) Baldwin[10] noted complete blindness in animals after the administration of large doses of quinine, but experimentation on animals specially directed to an elucidation of the pathogenesis of quinine-amaurosis begins with the research of H. Brunner,[11] and continues in the works of the author,[12] Paul Barabaschew,[13] De Bono,[14] and Ward Holden.[15]

**Etiology.**—In practically all cases of poisoning in human beings the drug has entered the system through the stomach. Blindness may follow the ingestion of any of the alkaloids of the cinchona bark, as the author has experimentally proved, and may even be caused by excessive doses of the tincture of the bark (Roosa), although naturally the salts of quinine are the most potent. That the drug itself and not a concomitant disease is the cause of the amaurosis is established by cases in which it was taken by mistake by healthy individuals (Giacomini), by experiments on animals and also on human beings (Barabaschew). The dose which may cause blindness varies from fifteen grains to one ounce in twenty-four hours, cases occurring under the influence of comparatively small doses

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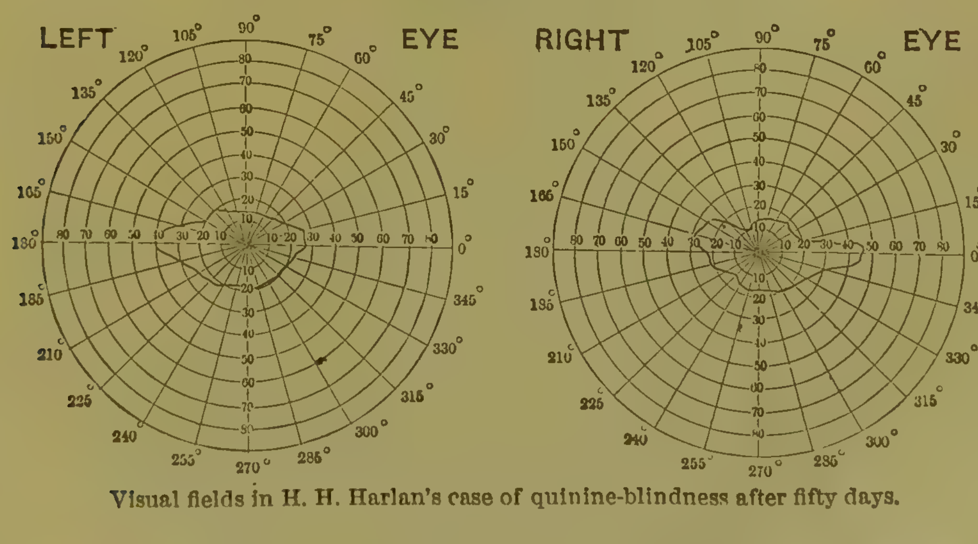
[1] Annali universali di medicina, Milano, 1829, vol. lii. p. 312.
[2] Schmidt's Jahrbucher, 1838, Bd. xx. S. 18.
[3] Ibidem, 1838, p. 292.
[4] Annali universali di medicina, Milano, 1841, vol. xcvii. p. 325.
[5] Western Medical and Surgical Journal, 1845, N. S., p. 396.
[6] American Journal of the Medical Sciences, 1847, N. S., vol. xiii. pp. 292–294.
[7] Graefe's Archiv, 1857, Bd. iii. Ss. 2, 396.
[8] Transactions of the American Medical Association, 1879, p. 411.
[9] Archives of Ophthalmology, 1879, vol. viii. p. 392.
[10] Loco citato.
[11] Ueber Chinamaurose, Inaugural Dissertation, Zürich, 1882.
[12] Transactions of the College of Physicians of Philadelphia, November, 1890; Transactions of the American Ophthalmological Society, 1891; The Toxic Amblyopias, Philadelphia, 1896.
[13] Archiv für Augenheilkunde, 1891, Bd. xxiii. S. 91.
[14] Archivio di ottalmologia, Palermo, 1894, vol. ii. pp. 171, 174.
[15] Transactions of the American Ophthalmological Society, 1898.

probably being due to idiosyncrasy. Of eighty-one cases which the author has gathered from the literature, forty-nine were males, twenty-five females, while in seven the sexes were not stated. The ages of the patients ranged from three to seventy-three years. The reports do not indicate that there is any racial immunity. According to De Gouvea, in Brazil, where much quinine is consumed, the affection is not uncommon. A highly neurotic temperament probably predisposes to this and other actions of quinine.

Symptoms.—In mild types of quinine-intoxication there may be temporary amblyopia without fundus lesions and diminished power of accommodation.

In severe cases the following symptoms may be present: blindness, complete or incomplete, usually developed with great suddenness and more absolute than in any other recoverable condition (Mellinger, Browne); dilatation of the pupils, absence of the light-reflex, imperfect response to accommodative effort, nystagmus, proptosis, occasionally divergent strabismus and increased intra-ocular tension (Tiffany); anæsthesia of the cornea and conjunctiva (Voorhies); extreme pallor of the optic disks and marked diminution in the size of the retinal vessels, simulating the appearances of advanced optic nerve atrophy; occasionally retinal haze and a cherry-colored spot in the macula, resembling embolism of the central artery of the retina (Gruening, Buller, Browne); gradually partial or complete restoration of central vision, associated at first with complete or partial color-blindness; later, slow renewal of the color-sense, which may ultimately return; often permanent diminution of the light-sense and color-sense and contraction of the visual field, the contraction usually assuming an elliptical shape; night-blindness (Swanzy); very exceptionally permanent blindness from optic nerve atrophy (Claiborne, Calhoun).

Fig. 13.




Visual fields in H. H. Harlan's case of quinine-blindness after fifty days.

Unusual phenomena are: normal ophthalmoscopic appearances (Garafolo, L. W. Fox); congestion of the retina and chorioid (Dickinson); primary ischæmia of the larger retinal vessels with overfilling of the retinal capillaries and those of the disk (Mellinger); and central scotomas (Jodko,

Galezowski). Although practically always a bilateral affection, one eye may be more affected and earlier than the other. One unilateral case (Graefe) is reported. According to Gruening,[1] "quinine-intoxication, associated with transitory amaurosis, may occur at times in a mild form with less pronounced contraction of the retinal arteries and veins and a decided fulness of the capillaries of the disk as a permanent condition."

**Diagnosis.**—This presents no difficulties, and depends upon the history of the case and the symptom-complex just detailed.

**Prognosis.**—In severe cases very exceptionally permanent blindness is the result, but taking all cases together, so far as improvement in central vision is concerned, even when the ophthalmoscopic picture is that of atrophy, the prognosis is good. But the period of recovery may be very protracted, lasting for months and even years, and, as before noted, defective vision may remain, and permanently imperfect color-sense and contracted visual fields are to be expected. Quinine-blindness once having occurred, relapses may be occasioned by small doses of the drug. (Knapp, Nettleship.)

**Pathology and Pathological Anatomy.**—The experimental researches of Brunner, Barabaschew, and the author indicate that the lesion in quinine-blindness is peripheral, that primarily there is an ischæmia of the retinal and optic nerve vessels caused by their intense contraction,[2] and that if the blindness continues sufficiently long atrophy of the optic nerve and optic tract (as far as they could be followed) ensues, associated with marked vascular changes characterized by vasculitis and endo-vasculitis, and finally obliteration of the lumen of the vessels.

According to the author's individual researches, the pathogenesis seemed to be somewhat as follows. In the earlier stages of the blindness—*i.e.*, from the first to the fourth week (ophthalmoscopically pallor of disks and great contraction of vessels)—the microscope reveals in the optic nerve imperfect differentiation of its fibrous trabeculæ and spreading apart of the individual nerve-fibrils, which are beaded and varicose and beginning to be atrophic. At this time no marked changes are present in the vessels. (Plate III., Fig. 2.) There is no evidence of neuritis. Later—*i.e.*, from two to three months after the onset of the blindness (ophthalmoscopically white disks and practically complete obliteration of vessels)—there are nearly complete atrophy of the optic nerve fibres (Plate III., Fig. 3), thickening and collapse of the nutrient arteries of the optic nerve, increase in the size of the perivascular lymph-spaces, and occasionally (two specimens) partial obliteration of the central vessel of the nerve with a partly organized thrombus. The atrophy extends throughout the nerve, chiasm, and tracts.

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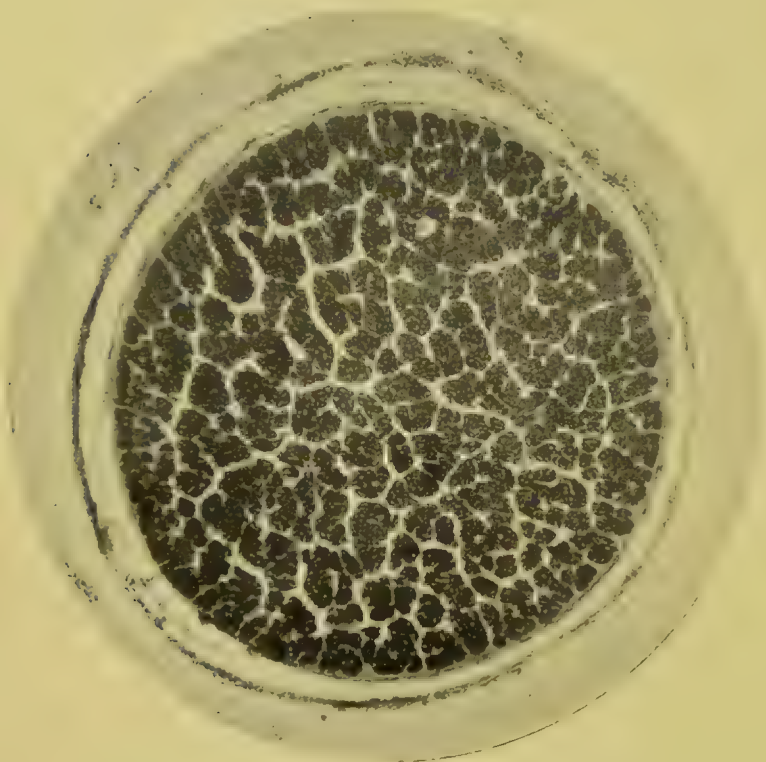
[1] New York Eye and Ear Infirmary Reports, 1897, vol. v. p. 6.

[2] Barabaschew believes that strong contraction of the peripheral blood-vessels is partly brought about by a condition of irritability of the vaso-motor centres caused by quinine. E. A. Browne suggests that it is caused by the local effect of highly cinchonized blood.

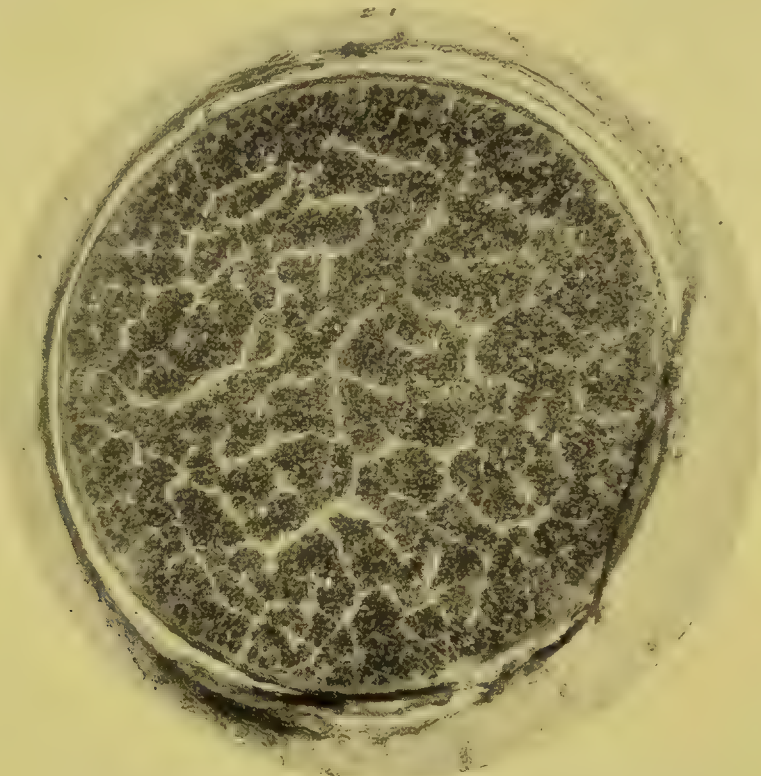
PLATE III.

Fig. 1.



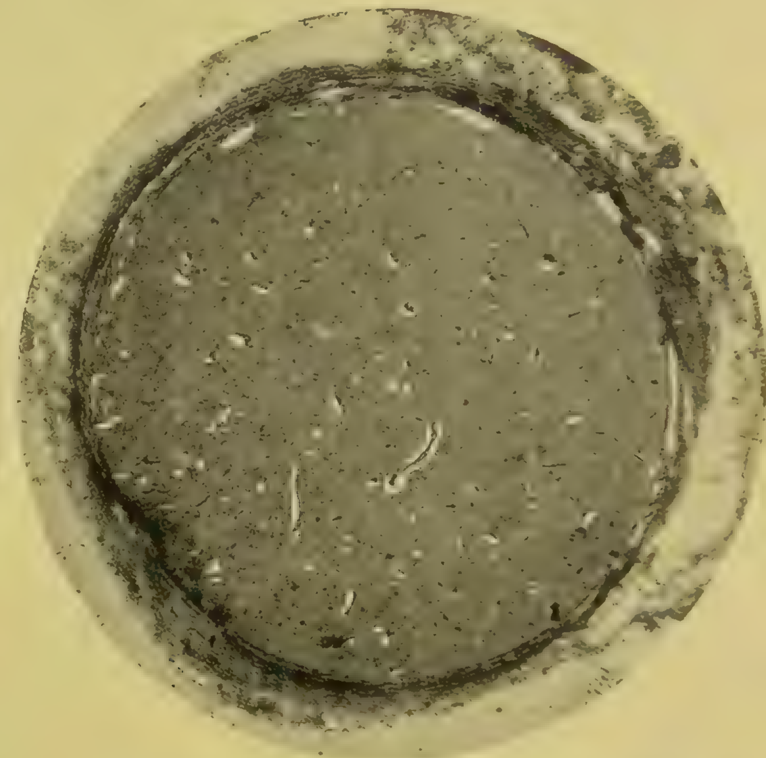
Normal optic nerve of dog. $\times$ 75. (Weigert.)

Fig. 2.



Transverse section of optic nerve of dog blind one month from quinine. Spreading apart of individual fibres which are beginning to be atrophic.

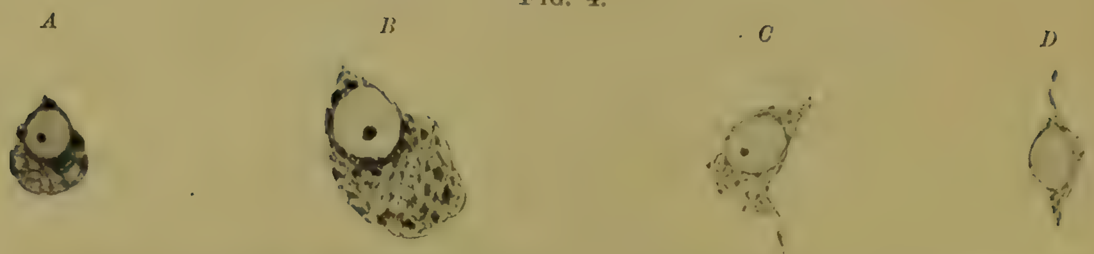
Fig. 3.



Transverse section of optic nerve of dog blind sixty-two days from quinine. Almost complete atrophy, with thickening and collapse of nutrient arteries. $\times$ 75.

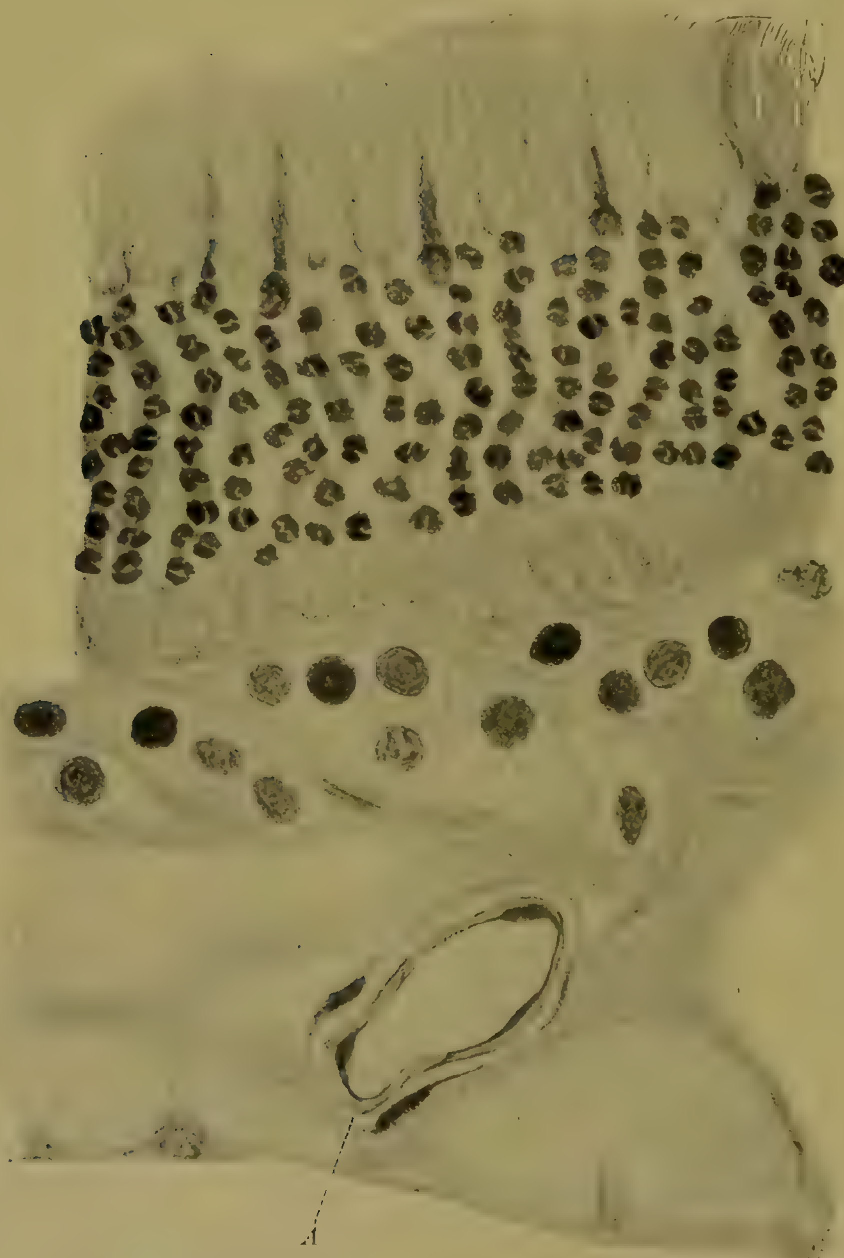
PLATE IV.

Fig. 4.




Ganglion cells in different stages of degeneration.—*A*, fine vacuolation; *B*, chromatolysis; *C* and *D*, breaking down of cell body. Third day of quinine-poisoning. (Holden.)

Fig. 5.




Almost complete absence of ganglion cells and nerve-fibres.—*A*, healthy artery. Sixth week of quinine-poisoning. (Holden.)

Even in sections of nerves which ophthalmoscopically give every evidence of atrophy, and which under the microscope show extensive atrophic changes, certain fibrils still remain unaffected, and, singly or in patches, stain somewhat imperfectly with the Weigert and Weigert-Pal reagents. This probably explains why vision is sometimes retained, even when the degenerative process appears to be complete. It is analogous to the retention of unaffected nerve-fibres within the atrophic areas of the papillo-macular bundle in cases of alcohol- and tobacco-amblyopia.

In some of the author's earlier quinine experiments alterations in the cortex of the visual area of the brain were discovered,—*i.e.*, shrinking of the cells and enlargement of the pericellular lymph-spaces,—but subsequent researches indicated that these lesions were probably due to some error in technique. It is probable that future investigations with the newer histological methods will show that there are really cortical changes under the influence of this poison, exactly as they have been found with other poisons, notably alcohol.[1]

The recent researches of Ward A. Holden[2] throw an entirely new light on the pathology of quinine-amblyopia. In this investigation, to quote Dr. Holden's own words, "A number of dogs were killed at periods ranging from two hours to seven weeks after the first injection of quinine, and the eyes, optic nerves, brains, and cords were examined by the Nissl-methylene-blue method for cell changes and the Marchi-osmic-acid method for nerve-fibre changes. Retinas examined on the third day, after several toxic doses had been given, revealed degenerative changes in a few ganglion cells (vacuolation, paleness and absence of chromophilic granules, and breaking down of the cell body) and changes in the nerve-fibres (a deposition in the nerve-fibre layer of large globules of a myeline-like character)." These degenerative changes gradually increased, and by the forty-seventh day the ganglion-cell layer and nerve-fibre layer had almost disappeared. The changes in the optic nerve were first noted on the seventeenth day, consisting in breaking down of the medullary sheaths of the fibres, and this degeneration of the nerve could be traced up to its fibres in the external geniculate body and pulvinar. In other words, again to quote Dr. Holden, "With the lessened but not obliterated blood-supply due to spasm of the vessels in quinine-poisoning, the less resistant elements of the inner layers—the ganglion cells and the nerve-fibres—break down, while the cells of the inner nuclear layer are not visibly affected. An ascending degeneration of the nerve-fibres follows the retinal changes." Dr. Holden failed to find histological alterations at this period in the retinal vessels or in those of the optic nerve. (Plate IV., Figs. 4 and 5.)

The experiments of Brunner and the author were performed at a time when the newer histological methods employed by Holden were unknown.

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[1] The older writers ascribed the blindness to an action of the drug on the cerebrum.

[2] Transactions of the American Ophthalmological Society, 1898.

It is now evident from his research that what they observed were the *secondary effects* of the toxic action of quinine, which probably *primarily* attacks the ganglion cells and causes the ascending degeneration of the optic nerve and tracts which has been described. The vascular changes which the writer particularly investigated would seem also to be secondary results, and it is probable that the conditions in the central vessel which he found, and ascribed to a thrombus, in the light of Holden's examinations must be regarded as a natural phenomenon in the central vein of the dog's optic nerve, and not one really due to the action of the drug. The gaps in our knowledge of the pathogenesis of experimental quinine-amblyopia have now been filled, with one exception,—namely, the cause of primary contraction of the retinal vessels, which creates the ischæmia, and which the writer and other authors have attributed to the effect of a highly cinchonized blood upon these vessels, an explanation, however, which is not entirely satisfactory.

De Bono, as the result of his researches, reaches entirely different conclusions in regard to the pathogenesis of quinine-amblyopia. According to him, the eyes of animals poisoned with quinine show only vascular changes in the chorioid and changes in the central vessels, but none in the retina and optic nerve. He believes that the ischæmia is not the sole cause of the functional disturbances, inasmuch as the latter may disappear while the ischæmia still remains, and ascribes the visual disturbance to alterations in the percipient end elements of the optic nerve, because in frogs poisoned by quinine there is lack of movement in the pigment molecules of the pigment epithelium. It is difficult to understand his failure to observe the atrophy which can always be produced in the optic nerve by quinine, except on the supposition that in animals his examinations were made before the period of atrophy sets in.

## AMBLYOPIA FROM OTHER ALKALOIDS OF THE CINCHONA BARK.

As already stated, any preparation of the bark is capable of producing the amaurosis which has been described in the preceding paragraphs. This has already been observed clinically, and has been proved by the author in experiments on animals.[1]

**Treatment of Quinine-Amaurosis.**—Necessarily the first caution to be observed is avoidance of further administration of any preparation of the cinchona barks. Inhalation of nitrite of amyl and the administration of digitalis represent rational modes of treatment. Strychnine was of service in some cases, notably in those associated with central scotoma, and galvanism has been advocated by Buller. If the intraocular tension is raised, as it was in Tiffany's case, eserine should be instilled. Other remedies which have been recommended are hydrobromic acid (L. W. Fox) and iodide of potassium (Calderai). There is some evidence to show that the administration of atropine produces favorable results in cases of quinine-poisoning.

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[1] The Toxic Amblyopias, pp. 199 and 200.

## AMBLYOPIA FROM SALICYLIC ACID.

According to Knapp, salicylic acid may create visual disturbances precisely analogous to those seen in quinine-blindness, although it would seem that there may also be blindness and mydriasis with normal optic papillæ. (Gatti.) The author has performed a number of experiments with salicylic acid analogous to those of Brunner and himself with quinine, and was able to produce blindness in dogs, which, however, was never complete. Ophthalmoscopically the disks were pale and the arteries small, with apparent contraction of the periphery of the visual field. In some cases there was an associated conjunctivitis, a symptom which has also been observed in human beings. The microscopical appearances which have been detailed in cases of quinine-blindness were, however, not found, probably because the poisoned dogs were not allowed to live for a sufficient length of time after the blindness began. There seems little doubt that the pathogenesis of the affection is the same as that of quinine.

## AMBLYOPIA FROM ASPIDIUM (U.S.P.), OR FILIX MAS (B.P.).

**History.**—Although the earliest report of the toxic properties of this drug appears in a monograph by Reinlein in 1812, it is only in comparatively recent times that its influence in originating visual disturbances has attracted attention and has been the subject of special investigation by two Japanese observers, Drs. Katayama and I. Okomoto,[1] who have collected twenty-three cases of poisoning, ten with temporary or permanent amblyopia of one or both eyes, five of them having occurred in Japan. Since this date a number of other cases have been reported in Germany, France, and Belgium, and experiments have been undertaken on animals, indicating that the visual disturbances are not confined to human beings. In other words, it is evident that the drug may cause marked amblyopia and even permanent atrophy of the optic nerve.

**Etiology.**—The cases of poisoning have followed the administration of the oleo-resin and also the liquid extract, and practically all of them have been the result of absorption of undue doses of the medicament when it has been administered as an anthelmintic. The size of the dose which has produced the toxic symptoms has varied considerably,—from three grammes of the extract up to very large doses.

**Symptoms.**—The ocular phenomena of filix mas amblyopia have not been well described, but have varied during the stage of blindness from negative ophthalmoscopic appearances (Baer, Schleier) to complete optic nerve atrophy (Grosz). Inouye describes yellowish-white radiating flecks in the macular region in a case of filix mas poisoning, but ascribes them to renal disorder brought about by the poison. Studies of the field of vision, the color-perception, and the light-sense are all imperfect.

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[1] The Sei-I-Kwai Medical Journal, 1892, vol. xi. pp. 101–121. For a larger collection, see Sidler-Huguenin, Correspondenz-Blatt für schweizer Aertze, September, 1898.

**Diagnosis and Prognosis.**—The diagnosis can be made by the history of the case. The prognosis is grave. In Sidler-Huguenin's collection of seventy-eight cases, twelve died and thirty-three suffered from permanent blindness.

**Pathology.**—Von Aubel ascribes the amaurotic effect of the male fern to its active crystalline principle, filicic acid, which he thinks acts through the vaso-motor nerves and produces contraction of the retinal arteries. According to Nuel, in a certain number of cases of male fern amblyopia in dogs there is blurring of the papilla, but microscopically no trace of an interstitial neuritis,—only an œdema of the nerve-head. This interstitial œdema is consecutive to the destruction of the nerve-fibres, and real neuritis, if it occurs, comes on later, but is neither interstitial nor retrobulbar. He believes that other cases of toxic amblyopia are of this nature, and that the original action of the drug is on the ganglion cells of the retina.

Masius[1] and Mahaim[2] have experimentally determined that the pathological process comprises an inflammatory condition of the capillary systems and secondary destruction of the nerve-fibres.

**Treatment.**—The treatment of this form of amblyopia does not differ from that detailed for other similar cases. Von Aubel particularly recommends nitrite of amyl and strychnine.

### AMBLYOPIA FROM SERPENT VIRUS.

Galezowski is authority for the statement of Armaral, of Brazil, that blindness after the bite of serpents is common. The author has been unable to find that this is one of the symptoms of the bite of the poisonous serpents of America. Congestion of the optic papilla and amaurosis lasting for months as the result of snake-bite have been reported by Laurençao, and S. Weir Mitchell has informed the author that he has seen retinal hemorrhage in experiments with serpent virus.

## II. POISONS WHICH PRODUCE AMBLYOPIA CHIEFLY BY CYCLOPLEGIA, MYDRIASIS, AND MYOSIS.

### AMBLYOPIA FROM THE MYDRIATIC AND MYOTIC DRUGS.

Whether true *atropine* or *belladonna amblyopia* occurs in the sense in which this term was used by the older writers (Himly, Beer, and others) is to be doubted. The amaurosis which they describe was no doubt the result of an action upon the ciliary muscle. What has been said of atropine is true of all other mydriatics,—hyoscine, daturine, duboisine, homatropine, scopolamine, etc.

Some of these drugs seem also to produce defective vision independently of their cycloplegic action. Duboisine, for example, may originate amblyopia and great narrowing of the visual field. (J. P. Worrell.) From the action of scopolamine there is sometimes a temporary amblyopia. (Pooley.)

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[1] Annales d'Oculistique, 1895, t. cxiv. p. 127.

[2] Académie royale de médecine de Belgique, 1896.

In *acute cocaine-poisoning* there may be maximum mydriasis and blindness owing to syncope, while temporary amblyopia and contracted retinal arteries have been observed after injection of the drug into the gum. (Schubert.)

Very interesting are the visual phenomena in *chronic cocaine-poisoning*, which may be summed up to be visual hallucinations, amblyopia, diplopia, chromatopsia, and hemianopsia, sometimes monolateral (Salis) and sometimes bilateral (Saury[1]).

*Amblyopia from the myotics*—that is, *eserine* and *pilocarpine*—may appear in the form of a disturbance of vision, caused by the contraction of the pupil and the approximation of the near and the far points. Temporary blindness in cases of general poisoning by physostigmine has in a few instances been reported without further particulars, and fugitive amblyopia is said to have followed injections of pilocarpine. (Fuhrmann.)

Two other myotic drugs—namely, *muscarine* and *curare*—may produce amblyopia by virtue of a myotic action and by causing cramp of the accommodation. Anæmia of the papilla is said by Galezowski to follow instillation of a collyrium of curare into the conjunctival cul-de-sac of rabbits.

### AMBLYOPIA FROM PTOMAINE-POISONING (BOTULISMUS, ALLANTIASIS).

Cases of poisoning in human beings dependent upon ptomaines and toxalbumins have usually followed the ingestion of spoiled meat, fish, sausage, cream, pastry, etc. As many of the ptomaines are basic compounds closely simulating the vegetable alkaloids,—for example, atropine, etc.,—it is not unnatural that the ocular symptoms would be analogous to those produced by the vegetable drugs which these substances resemble in physiological action. In general terms, they are dim vision or transitory amblyopia, bilateral *paresis of accommodation with or without mydriasis*, negative ophthalmoscopic appearances, and frequently palsies of the external ocular muscles. Indeed, the most interesting ocular lesion in connection with ptomaine-poisoning centres around the paralysis of the ocular muscles, and is therefore not specially germane to the present subject. There may be simply ptosis, palsy of a single ocular muscle, or complete ophthalmoplegia externa.

## III. POISONOUS SUBSTANCES WHICH PRODUCE VARIOUS OCULAR PHENOMENA, BUT NOT AMBLYOPIA IN THE USUAL ACCEPTATION OF THIS TERM.

A certain number of drugs and chemicals usually included in the lists of substances capable of producing toxic amblyopia have no special claim to such a position, unless some of them should be accorded a place among

[1] Annales médico-psychologiques, 1889, t. ix., 7 s., p. 439.

the agents which cause defective vision by mydriasis. The following are examples of this class:

Benzine (petroleum ether), which in poisonous doses causes immobile pupil and nystagmus; sulphonal, which, like bromide of potassium, may produce ptosis;[1] trional, which after excessive doses may originate tinnitus and muscæ volitantes; aconite, which causes mydriasis and other visual phenomena dependent upon its depressant action upon the circulation; pink-root (the root of the *Spigelia marilandica*), which resembles the mydriatics in its toxic symptoms, and therefore blurs vision by mydriasis; pomegranate, which is said to create retinal congestion; piscidium (Jamaica dog-wood), which is a mydriatic when its extract is freely administered; cytisine (the active principle of the *Cytisus laburnum*), which, according to Albutt, in toxic dose dilates the pupil, blanches the optic papilla, and contracts the retinal vessels; sweet spirits of nitre, which in poisonous quantities causes dilated and motionless pupils; esculin (horse-chestnut), which originates symptoms similar to belladonna-poisoning; menthol, which, like naphtalin, is said to produce lenticular opacities if it is pushed to the lethal issue in animals; sulphur, which in one case is said to have caused mydriasis, exophthalmos, and anæsthesia of the cornea; sulphuric acid, which may originate an acute ophthalmoplegia externa, due to hemorrhagic polioencephalitis; oxalic acid, which creates visual disturbances (mydriasis) as the result of the exhaustion which follows upon its acute toxæmia; nitrous oxide, which, according to Aldridge, causes dilatation of the retinal arteries and increased redness of the papilla; and light carburetted hydrogen gas (fire-damp), which by some authorities is held responsible for miners' nystagmus.

Strabismus and exophthalmos, according to Kepler, have been caused by the poisonous action of saponin, while anæsthesia of the cornea, as well as clouding of its structure and consequent depreciation of vision, may be brought about by the instillation into the conjunctival cul-de-sac of a two per cent. solution of apomorphine and of strophanthin and ouabœine. Erythrophlœine (Haya-poison) anæsthetizes the cornea and irritates the conjunctiva. Chrysarobin and podophyllin, especially the dry roots of the latter drug, may cause an active conjunctivitis.

*Amblyopia from the anæsthetics*, particularly ether and chloroform, if Reymond's case be excepted, through an action of the papillo-macular bundle, apparently does not occur. They may bring about anomalous pupillary reactions, and occasionally have caused detachment of the retina. Chloride of ethyl affects vision by creating opacity of the cornea. (Dubois and Roux.)

Toxic Chromatopsia.—A prominent visual phenomenon under the poisonous influence of certain drugs is colored vision, usually xanthopsia,

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[1] It is possible that future investigations will show that the ocular symptoms of this drug are more pronounced than have thus far been reported.

or "yellow seeing." Of the various drugs which may produce this phenomenon *santonin* is the most notorious, and the phenomena have been most carefully investigated by Rose, Knies, and other observers. A deep yellow tint is imparted to everything in the range of vision; sometimes yellow is replaced by green, and it is stated that the tint may be red or blue. According to Knies, the yellow seeing appears in from ten to fifteen minutes after the toxic or full physiological action of the drug sets in, and is preceded by a period of "violet seeing." Ophthalmoscopically there are no changes, and the central vision and the pupillary phenomena are normal. Retinal adaptability, however, is diminished. Other drugs which produce this symptom are nitrite of amyl (Peck, Landendorf), picric acid, chromic acid, when it is used as a local application, digitalis[1] (Jeanton), carbonic oxide (Hilbert), and tobacco (Simi and the author). Cannabis indica may cause violet vision (Werner), and coffee red vision.

TOXIC HYSTERIA IN RELATION TO TOXIC AMBLYOPIA.—Among the workers in lead-factories the so-called hysterical lead-paralysis is not uncommon. Those affected suffer from hemianæsthesia, amblyopia on the same side, anosmia on the opposite side, dyschromatopsia and reversal of the normal sequence of color-perception.[2] A similar reversal in the color-lines has been noted in nitrobenzol-poisoning. Among other toxic agents which are supposed to be capable of originating an hysterical condition may be mentioned alcohol, mercury, and bisulphide of carbon. The subjects may suffer from hemianæsthesia, hemiplegia, restriction of the visual field, etc., and may be cured by suggestion or by remedies of indifferent potency. Many of the well-marked hysterical stigmata can be found at times in subjects of chronic alcoholism.[3]

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[1] During poisoning with this drug there may be mydriasis and nebulous vision, and L. Lewin (Die Nebenwirkungen der Arzneimittel, Berlin, 1881, S. 191) mentions among the toxic symptoms attributable to this drug amblyopia and double-sight. This amblyopia has also been ascribed to *digitoxin*.

[2] Pontain, Recueil d'Ophtalmologie, 3 s., 1887, t. ix. p. 620.

[3] Goinou, Les agents provocateurs de l'hystérie, Thèse, Paris, 1889. In this interesting thesis the entire literature of this subject is reviewed. Recently the author has observed hysterical amblyopia associated with toxic amblyopia, apparently due to the excessive use of tobacco. (Ophthalmic Record, January, 1899.)

# THE ENTOZOA OF THE HUMAN EYE.

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As the natural history of intestinal worms in general is of comparatively recent date, so is also our knowledge of the diseases caused by entozoa in the human eye. The first observation on the cysticercus was made by Schott in 1830 (cysticercus in the anterior chamber). The most important of these affections, cysticercus in the posterior segment of the eye, was first discovered in 1853 by von Graefe. Of a much earlier date is the knowledge of echinococcus of the orbit, although its animal nature was not fully established until the present century. The clinical significance of all these diseased conditions lies principally in the inflammatory reaction which the parasite causes; seldom does it become injurious by reason of its size. These diseases are also instructive from a zoological stand-point, because they give a clear insight into the life-history of organisms which otherwise flourish concealed in the deep tissues of the body.

The eye with its immediate surroundings serves as a place of residence for many forms of entozoa, of which the cysticercus cellulosæ is the principal, not only because it is most frequently met with, but also because it may be found in any part of the eye or its appendages. Other varieties of entozoa are found only in special regions. Finally, many of the entozoa are so rare that they are of no consequence either to the practitioner or to the general pathologist.

### CYSTICERCUS CELLULOSÆ (PORK MEASLES).

**Natural History.**—The ripe segments of the tænia solium Rudolfii inhabiting the human intestine pass off either alone or with the stools; they are completely filled with eggs, each containing a fully developed embryo in a thick porous capsule. These eggs, or under certain circumstances whole segments, enter the stomach of the hog with the food, where the capsules are digested and the embryos set free.

The free embryo appears as a small spherical body from 0.022 to 0.028 millimetre in diameter, and armed with six hooks; with their aid it perforates the intestinal wall and either migrates independently farther into the loose cellular tissue, or is carried by the blood- (or lymph-) stream to remote parts of the body. After it has effected a lodgement in some portion of the vascular system, it penetrates into the tissues and throws off its embryonal hooks. It is soon enveloped in a capsule, which consists first of cells, later of connective tissue; within this the further development of the parasite takes place. Immediately after the throwing off of the embryonal hooks the young cysticercus appears as a solid spherical body, which grows rapidly and is very soon converted into a small cyst by accumulation of fluid. Its original tissue is thereby crowded towards its periphery and there becomes differentiated into three layers. The external layer is composed of a structureless membrane with warty projections, the middle layer of numerous spindle cells, and the innermost layer of a loose cellular tissue traversed by muscular fibres. When the cyst has attained a diameter of about one millimetre the first rudiments of the head of the future worm are discernible.

This appears as a bud like projection on the inner surface of the cyst, and is formed by proliferation of the various layers composing the cyst-wall. As this bud grows it penetrates still farther inward, causing a tube-like invagination of the wall of the cyst. By further differentiation this invaginated process forms the head, neck, and body of the worm, and also a muscular sac—the receptaculum—enclosing these structures. The head is developed at the bottom of this tubular invagination, the circlet of hooks and the suckers nearer its orifice and directed towards its cavity. When by a process of evagination the head is extruded, the inner lining of what was a hollow tube becomes the outer covering of a projection, and the organs that were at first directed towards the cavity of the former, now present externally upon the latter, but in a reversed order, the rostellum that originally was at the bottom now being at the top.

The fully developed head may be extruded at pleasure, and in old cysts is usually so found. At its extremity a short snout-like projection, the rostellum, encircled by a ring of from twenty-six to twenty-eight hooklets, is seen, and below this four sucking-disks. Under this we find the narrow neck, joining the somewhat broader, transversely wrinkled body, which merges with a slight constriction into the caudal cyst. The head, neck, and body are white, although in very old specimens the sucking-disks are pigmented. The cyst always retains a certain degree of transparency. Two and a half months usually elapse before the head is completely developed, and after this no material change takes place, except that the cyst becomes larger and the body increases in length. The life of the cysticercus is estimated at several years.

**Etiology and Distribution.**—Through the ingestion of raw or imperfectly cooked pork one can acquire only the tænia solium; from this, infection with the measles may result. It is indispensable that the ripe egg

be deposited in the stomach, as the embryo can escape only after its envelopes are digested. The egg or the entire ripe segment may find its way into the stomach in one of two ways, either directly from the intestine through antiperistaltic motion or from without by the mouth. By the first method only the host of the tapeworm, by the second other individuals, may be infected. The circumstances under which this infection takes place are manifold: river water contaminated by human fæces, vegetables grown in freshly manured fields, or filthy hands may communicate the contagion.

The independent motility and spontaneous discharge of the fertile segments, as well as such close intercourse as the use of common lodgings or taking meals with a person who is suffering with tapeworm, especially if both parties are not particular as to cleanliness, give the opportunity for such infection. Much stress was formerly laid upon the latter mode of infection for the reason that cysticercus was often found in those subjects who at the time were apparently not suffering from tapeworm. It has since been shown—a point to which Hirschberg calls especial attention—that the parasite may easily be overlooked when there is no spontaneous escape of segments. Attempts at dislodgement of the worm are in almost every instance successful. It is not to be wondered at that the poorer classes suffer more from cysticercus than the wealthy, although among the latter the disease is not infrequent. Infection by cysticercus is most common between the ages of twenty and thirty (Hirschberg, Germelmann). According to Hirschberg, both sexes are equally affected, while von Graefe says that it is more common in the male. The cysticercus cellulosæ can naturally occur only where tænia solium is found, although the areas of distribution do not always coincide. For instance, tænia solium is very frequent in Switzerland, where the cysticercus cellulosæ is very rare. In England, France, Belgium, Southern Germany, Austria, and Russia the cysticercus cellulosæ is a rarity. It seems to be more prevalent in Portugal, and a considerable number of cases have been reported from Italy. Without doubt it is found with greatest frequency in Northern Germany, where, according to von Graefe, it occurred in the ratio of one to one thousand in eye patients, while Hirschberg reports it to be at times in twice this proportion. Hirschberg's latest statistics show that a gratifying diminution of the disease has been noticed even in Berlin. Indeed, in recent years the disease is said to have entirely disappeared.

The immigration of the embryo into the eye occurs passively by way of the arterial blood-stream. In this way it reaches some point in the retinal, uveal, or conjunctival vascular system, where, on account of its size, it becomes arrested. This point is generally reached before the capillary system begins. There are, however, capillaries in the choroid wide enough (0.036 millimetre) to allow the passage of the embryo. Still, it may get into comparatively narrow vessels, as the following shows. The arteria centralis retinæ is 0.2 millimetre in diameter at its narrowest point before it enters the lamina cribrosa; its diameter is therefore at least seven

times greater than that of the embryo (maximum 0.028). Consequently, it cannot be conceded that the embryo will produce phenomena of embolism of a principal vessel on entering the retinal vascular system, as has been supposed by several observers. It would be more likely to lodge in one of the smaller vascular twigs, producing branch embolism with but slight functional disturbance. The embryo is swept more frequently into the posterior ciliary arteries than into any of the other independent vascular systems of the eye, as this system receives the largest amount of blood and has the greatest number of afferent branches.

Although the cysticercus is often found in numbers in the skin and brain, in the eye it is almost always solitary. In only a few cases have two cysticerci been found in one eye (Kauweki, under the conjunctiva; Becker, Gradenigo, Cohn, and Alfred von Graefe, in the posterior segment); the occurrence of three subretinal encapsulated cysticerci in one eye, which Schöbl has lately reported, must be regarded as extremely uncommon; almost equally rare is the simultaneous occurrence of cysticerci in the eye and in the skin (Schultz, Hirschberg) or in the brain (von Graefe, Hock, Sasson Alcalaï).

**Course.**—The symptoms vary greatly, according to the location of the parasite. But it is not so much the presence of the worm, as such, as the reaction it occasions that determines the course of the disease. The reaction of the tissues against the parasite lodged in them is manifested by the symptoms of chronic inflammation with a proliferation of connective tissue, rarely terminating in suppuration. This must be regarded as the consequence of irritation produced by the development of the parasite, and in no way due to micro-organisms (Leber), as the removal of the parasite stops the inflammatory process. In the case of the intra-ocular cysticercus, which is best known, and whose symptoms obtrude themselves upon the patient persistently and early in the disease, the reaction may be divided into two more or less distinct periods. The first period, the *initial reaction* or the *invasion-inflammation*, follows immediately upon the immigration of the parasite. It is transient, and harms neither the parasite nor the affected organ. Following this is a more or less distinct *stage of indolence*, during which the parasite continues to thrive. At last, however, a reaction of gradually increasing intensity occurs, which in time leads not only to loss of the eye, but also to destruction of the parasite. This may therefore be denominated the *terminal reaction*. The duration and intensity of these stages are quite variable; there may be cases in which the stage of indolence is absent. It must not be forgotten, however, that in the majority of cases the patient does not seek medical aid until the beginning of the terminal reaction, as this stage, through its duration and the intensity of its phenomena, makes itself much more noticeable than the initial reaction. The parasite shows a disposition to grow and wander in the direction of least resistance; consequently it soon arrives in loose or soft tissues (subconjunctival tissue or the vitreous) or in spaces filled with fluid (anterior

chamber). These changes of place are not due to the worm's own movements, which are not of a character to produce them. They result rather from the mere increase in size of the cyst, and the changes occurring in its vicinity, such as exudation, contraction of the capsule, etc.

THE EXTRA-OCULAR CYSTICERCUS is of slight importance. Its seat is the loose connective tissue in the neighborhood of the eyeball and its appendages; the disturbances it creates are slight, and, above all, vision is not impaired. As in subcutaneous tissue generally, the cysticercus may also develop under the skin of the lids. It there appears as a small, rounded, tense tumor, movable under the skin and over the tarsus. The orbit harbors the cyst only in its anterior segment, close to the edge of the orbit, and even here it is but rarely seen. The inflammation it produces in this location is so intense that the symptoms have some resemblance to those of an orbital phlegmon of limited area. Fromaget calls attention to the paroxysmal character of these inflammatory attacks and their frequent recurrence. There is no exophthalmus, and lateral dislocation is rare. The cysticercus is more prevalent in the subconjunctival connective tissue than in the places named. Here it may occupy the most variable positions, preferring, however, the peripheral portions of the ocular conjunctiva. As a rule, its development is not noticeable until it is made apparent by the size of the tumor. The capsule of the parasite is composed externally of a layer of connective tissue, internally of a stratum of granulation tissue, with a greater or less number of giant cells, and is often firmly adherent to the tendon of one of the recti muscles. Small quantities of pus are not infrequently present, but they are always seen in the immediate vicinity of the cyst. The tumor attains about the size of a pea, and, if the capsule is delicate, appears as a translucent cyst, in which the cephalic bud is indicated by a white spot. In this case the diagnosis is easy, as all other cysts of the conjunctiva are perfectly clear vesicles. When the capsule is dense, the parasite itself is invisible; the tumor is of a reddish color and seems solid, and may readily be mistaken for a neoplasm; the diagnosis can be made only by a consideration of the short duration of the process, the accompanying inflammation, and the seat of the swelling. True neoplasms prefer other situations. Lodgement in Tenon's space may be mentioned as an extremely rare occurrence.

THE INTRA-OCULAR CYSTICERCUS is of the greatest importance, as the inflammatory reaction leads to severe visual disturbance. The various regions of the eye occupied by the cysticercus must receive separate consideration.

*Anterior Chamber.*—The invasion occurs through the blood-vessels of the iris, and the initial reaction presents the symptoms of an iritis (invasion-iritis). The growing parasite very soon finds its way into the anterior chamber, where it may remain for a long time without producing material disturbance.

In this, the stage of indolence, it is, as a rule, found floating free in the aqueous humor or adherent to one of the walls of the anterior chamber,

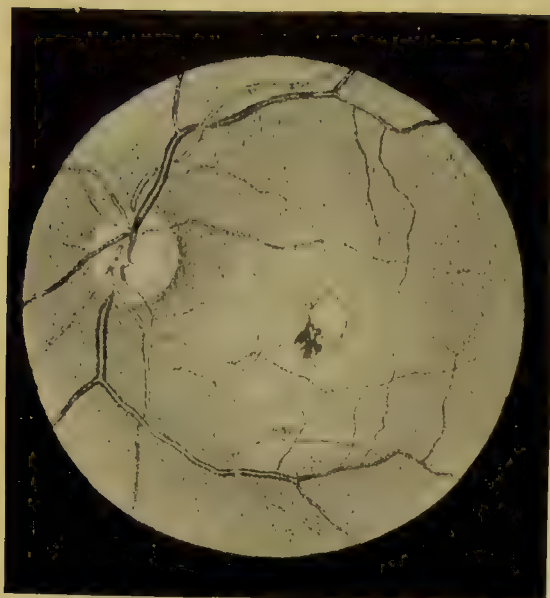
sometimes the iris, sometimes the posterior surface of the cornea. Here the cyst has a characteristic appearance (compare, below, subretinal and vitreous cysticercus). Motion is excited in it by the action of light, though whether directly or indirectly in consequence of the movements of the iris induced by it is undecided. Atropine is said to paralyze the cysticercus as well as the sphincter muscle of the iris. During this time the subjective symptoms are slight, the degree of the visual disturbance varying according to the location of the cyst. An exudative iritis develops after the affection has lasted some time, as the terminal reaction, and leads to the formation of adhesions between the iris and the parasite, and ultimately to the loss of sight.

In the *posterior chamber* the cysticercus has been observed in but one case (von Hasner); a segment of the cyst projected over the pupillary border. There is also but one instance known of cysticercus in the lens (von Graefe); a cysticercus presented itself immediately upon delivery of the lens in a case of seemingly uncomplicated cataract.

Cysticercus in the *posterior segment of the eye* (under the retina and in the vitreous) is of the greatest clinical as well as therapeutical importance. In regions where this disease is prevalent, it is more than eight times more frequent in this portion of the eye than in all other parts together. These two locations must be considered together, as cases occur where the parasite has occupied both positions successively. The invasion may occur through the choroidal vessels or through those of the retina; the first way is certainly the more frequent. Invasion by way of the retinal vessels is conceded by some authors to be a possible, though a rare occurrence; and by others (as lately by von Graefe) it is denied. I have observed a case in which the clinical peculiarities seem to justify the theory of such an invasion. I refer to remarks on page 845 as to the possibility of such an occurrence.

The initial reaction usually presents the symptoms of a uveitis (sometimes of an iritis), more rarely those of retinitis without participation of the uveal tract (Jany, Salzmann). These differences may be easily explained by the various modes of invasion. In both instances local disturbance of circulation must occur, which, owing to the peculiar vascular distribution in the retina, is most noticeable there, and impresses a hemorrhagic character upon the inflammation. This invasion inflammation extends over a comparatively large area of the affected membrane, but subsides after a few days, leaving no changes except at the spot where the parasite has lodged. If this be in the choroid, it will there pass through the first stages of its development, which cannot be then observed. According to the description of von Graefe, there first appears a bluish-gray opacity of slight extent, which rapidly enlarges, and gradually rises above the level of the neighboring retina. Later the small cyst emerges from the opacity, and either enters the vitreous at once or is visible under the retina.

Fig. 1.



A recently immigrated cysticercus; diameter 0.5 millimetre. The central cavity is not yet formed; the retina is slightly detached and clouded in spots. (Invasion-retinitis.)

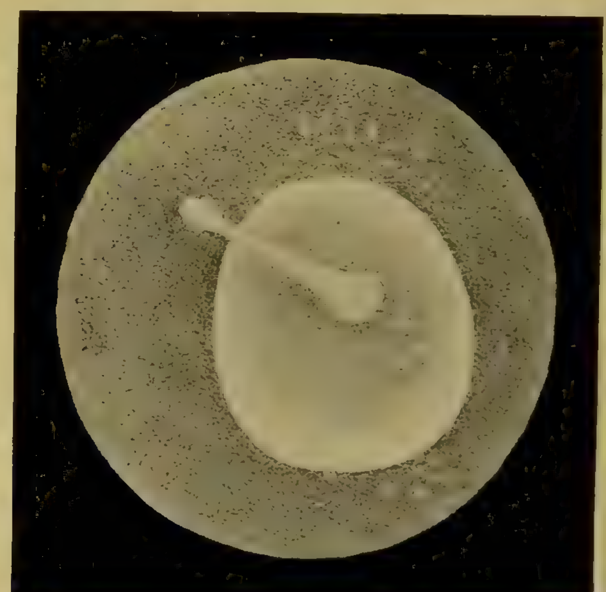
Fig. 2.

Same as Fig. 1, eight days later. The parasite is now two millimetres in diameter and appears as a cyst. The first traces of the cephalic bud are visible; the surrounding retina is normal. (Stage of indolence.)

Fig. 3.

Older cysticercus, five millimetres in diameter, with well-developed head. (Stage of indolence.) After Fuch's Lehrbuch der Augenheilkunde, 3d ed.

Fig. 4.



Cysticercus free in the vitreous. After Liebreich's Atlas der Ophthalmoskopie, Taf. VII.

If the invasion occurs through the retinal vessels, the parasite is more accessible to direct observation, and the earlier stages of its development may be followed with the ophthalmoscope. A cysticercus of about 0.5 millimetre diameter (one-third of the diameter of the papilla), which, judging from the occurrence of visual disturbance, was five days old, appeared as a bluish-white, opaque, roundish body, and showed lively movements. The surrounding retina was clouded in spots over a considerable area and was somewhat detached (invasion-retinitis: see Fig. 1). Three days later the parasite was about the size of the papilla, and presented the appearance of a very delicate, almost transparent vesicle. Four days after this the last vestiges of the invasion-retinitis had disappeared, and the young cysticercus, on which the first traces of the cephalic bud were already visible as a small whitish spot, lay wholly free under the otherwise normal and transparent retina. (See Fig. 2.)

At this point the disease had entered the stage of indolence, the parasite growing slowly under the retina without producing other lesions than the detachment and the visual disturbance necessarily connected therewith. The presence of a sharply defined retinal detachment of a hemispherical or hemi-ellipsoidal form without folds upon its surface is characteristic of this stage. The detached retina is perfectly transparent; the vessels appear normal, although the descending portions are a little darker, as the vessel-reflex is absent. The cyst, somewhat flattened, completely fills the space between the retina and the choroid. Its edges are whitish, the centre transmitting the red color of the fundus without its details, as through a delicate gray veil. The movements of the cyst resemble the peristaltic action of the intestines, and produce manifold changes in its shape. At one point, usually towards the retina, in the centre of the cyst, the cephalic bud is visible as a faint white spot whose form and location frequently change. (See Fig. 3.)[1] Sometimes a light stripe, which is assumed to be an abrasion of the pigmented epithelium caused by the parasite's wandering, is seen running towards the cyst. The movements of the parasite under the retina are seldom observed at the time, although the resulting change of its location and the streak before alluded to are seen often enough. Mitvalsky had an opportunity of witnessing such a migration, and describes it as follows: "A circumscribed projection separated from the rest of the cyst by a deep furrow made its appearance on one side. This projecting process seemed to be more delicate in structure than the original cyst, and was in active motion, while the older part of the cyst remained quiet. In proportion as the projecting part increased in size the volume of the old cyst diminished as though its contents were being emptied into the projecting part." During the stage of indolence the subjective symptoms are restricted to the presence of a circumscribed scotoma which corresponds in location and extent to the retinal detachment. The nearer the seat of the parasite to the macular

[1] Liebreich, Atlas der Ophthalmoskopie, Taf. VII. Fig. 6.

region the more apparent is this blind area. When peripheral it may altogether escape the patient's observation, so that the parasite is discovered only by accident (Hirschberg, Stoelting). The movements of the cyst often occasion photopsia.

After a longer or a shorter period the disease enters the stage of terminal reaction (true cysticercus inflammation of Leber). Probably one of the earliest signs of this is an increase in the extent of the retinal detachment. While in the indolent stage the retina is in close contact with the parasite, a serous exudation now takes place beneath this membrane, so that the parasite lies in a fluid-filled space. For this there are various causes. The movements of the cyst and its efforts at extruding its head, its migrations under the retina, and the inflammatory reaction in the neighboring choroid, either directly by effusion under the membrane or indirectly by structural changes in the vitreous, aid in producing this serous exudation. About this time opacities of the vitreous develop. These are delicate and of slight extent at first, but soon become characteristic. The mobility of the cyst is of course increased by this serous retinal detachment, and it assumes its normal ellipsoidal shape. The extrusion of the head, hitherto invaginated, is now possible.

At this time the ophthalmoscopic appearances are those due to serous detachment of the retina and the presence of the cyst, whose subretinal location is shown in the upright image by the fact that the retinal vessels lie in front of it. The cyst is now less distinct than in the preceding stage, as the vitreous opacities, in addition to the superjacent retina, obscure the view. In many cases the parasite remains permanently under the retina, and encapsulation ensues through gradual increase of the inflammation. It is rarely possible to observe this capsule with the opthalmoscope. When visible it is opaque and of a yellowish color (Jakobson), occasionally presenting a silky sheen at certain points (Stoelting). The capsule is rarely developed to such an extent as to simulate the appearance of an intra-ocular tumor. In most cases of this kind the inflammation in the anterior segment is so far advanced that ophthalmoscopic examination is impossible, but anatomical investigation of the enucleated bulbus shows this condition to be relatively frequent.

As a rule, the stage of serous detachment of the retina is followed by the passage of the parasite into the vitreous. At first the head penetrates the retina and projects into the vitreous, the cyst still remaining under the retina,[1] but after a few days the parasite passes altogether into the vitreous. By this change of position the cyst is brought nearer the lens, and must therefore appear smaller than in its former subretinal location. In so far as it is not obscured by opacities in the vitreous it is now seen with great distinctness. (Fig. 4.) Its edge appears as a colored zone which in smaller specimens is yellowish, in larger ones is of a luminous red. The move-

[1] Jaeger, Atlas, Taf. XVIII. Fig. 83.

ments of the cyst take place in the same way as in the subretinal variety, except that here locomotion of the entire cyst may sometimes be observed. The special feature of a cysticercus of the vitreous is the play of the head: at one time it is retracted and again partially or wholly extruded. All the details described on page 844 may be recognized on the extruded head, which describes circular movements about its insertion in the caudal cyst or may at times be seen either rigidly extended or pendulous. The rostellum and the individual sucking-disks are likewise seen sometimes extruded and sometimes retracted. When the head is fully drawn in, its point of junction with the cyst resembles a slit. It is surrounded by radiating folds, and contracts and expands as if by the aid of a sphincter. In this manner the movements of the parasite present an ever-changing and interesting spectacle.

During prolonged observation, locomotion of the cyst, either spontaneous or due to the position of the patient, may be frequently observed. This free mobility of the cyst is of unfavorable prognostic importance as regards an operation. In other cases it is fixed by bands in the vitreous,—*i.e.*, it occupies permanently the same place near the ocular wall. The chances for the success of an operation are more favorable in these cases. During this stage the vitreous is never completely clear. Rounded opacities resembling the precipitates on the posterior corneal surface are frequently found in the immediate neighborhood of the parasite. The vitreous is, moreover, permeated by a system of communicating veil-like opacities of relatively slight mobility and of such a characteristic appearance that the expert may from them alone be led to suspect the presence of a cysticercus. This symptom is of considerable importance, as with the increasing opacity of the vitreous the visibility of the parasite constantly decreases, so that at last a particularly bright reflex alone indicates its seat.

The original location of the parasite remains visible even after its rupture into the vitreous. It appears as a glaring white cicatrix permeated by atypical vessels, and may on superficial examination be easily mistaken for a second cyst. The detachment of the retina keeps pace with the progressive degeneration of the vitreous, and in the later stages becomes total. These various changes lead to serious impairment of vision. With the occurrence of the serous retinal detachment the originally small scotoma increases and gives rise to a large defect in the field of vision. Central vision likewise suffers through this as well as in consequence of the opacities in the vitreous. After the cyst has penetrated into the vitreous it may happen that its shadow falls upon parts of the retina still functionally active, and an entoptic image of the parasite is thereby produced. This image appears as a roundish shadow in a point of the visual field diametrically opposite to the location of the parasite. The shadow may move and change in form, and occasionally show a conical projection, the shadow of the head. The cysticercus does not perhaps attain the same size in the eye as in other organs, but its development is always typical. In one isolated

instance (de Vincentiis) has an anomaly in the form of the parasite, the so called *cysticercus racemosus*, been observed in the interior of the eye. Such anomalies, as is well known, are frequent in the brain.

With the progress of the disease vision gradually diminishes until total blindness results. Up to this time pathological changes have taken place only in the posterior segment of the globe. At last, however (from one-half to two years after the immigration of the embryo), the terminal reaction becomes so intense that symptoms of inflammation are discernible in the anterior segment, and an insidious irido-cyclitis attended by redness and pain develops, which at last leads to opacity of the lens and atrophy of the globe. The condition of the eye then strongly resembles that at the termination of traumatic irido-cyclitis, and often warrants the fear of sympathetic inflammation in the other eye. No authentic case of sympathetic inflammation originating from cysticercus is, however, known.

The anatomical changes, apart from the presence of the parasite, are those of chronic inflammation of the uveal tract, with its consequences, cataract, degeneration and detachment of the retina, and atrophy of the globe. The inflammation may even extend to the sclera (Wagenmann). The cysticercus is surrounded by a capsule composed externally of connective tissue and internally of granulation tissue. If the location of the cyst be favorable, the internal coats of the eye may participate in its formation. More or less purulent exudation and frequently giant cells are found in the immediate neighborhood of the parasite; the latter resemble the giant cells of tubercle to such an extent that the entire inflammatory reaction has been mistaken for tuberculosis (Schroeder).

Even in eyes that have become blind the parasite may retain its vitality for a long time. Living cysticerci have been found several times in enucleated eyes after the disease had existed two years. In one case the cyst was but recently dead, although the disease had lasted for ten years (Saemisch). As a rule, the parasite dies after several years, in consequence of the inflammation provoked by its own presence. After this the giant cells appear to bring about its absorption (Schroeder).

**Diagnosis** of the intra-ocular cysticercus is very easy if the parasite can be plainly seen. It becomes difficult only in advanced cases, as then opacities in the vitreous or beginning inflammation in the anterior segment obscure the view. Experience is required in order to justify a positive diagnosis from the form of the opacities in the vitreous. The demonstration of a rounded cyst, clearly defined on all sides, greatly increases the probability of a correct diagnosis. For this, examination with a strong convex lens (20 D) by the inverted image is recommended. The lesser enlargement of the ophthalmoscopic picture facilitates the search by permitting the examination of a large portion of the fundus at one time. An absolutely certain diagnosis can be made only when the intrinsic movements of the worm are recognized.

While it is very easy to observe the movements of a cysticercus when

free and fully visible, it may become very difficult when the greater part of the cyst is obscured by vitreous opacities. In such cases it is well to examine with the upright image without correction at a considerable distance, so that the cyst and the affected eye may be observed simultaneously. The eye should be in such a position that only a small segment of the cyst is visible through the illuminated pupil. The examiner now carefully examines the relation of the edge of the cyst to the pupil. If while the eye is absolutely quiet the relative position of the cyst to the pupil changes, the diagnosis is established. Care must be taken not to confound communicated with intrinsic movements, nor must it be forgotten that any formation suspended in the vitreous may be caused to vibrate by motions of the eye, and that these vibrations may continue for some time after the eye has become quiet. In some cases continued observation and repeated inspection may be necessary to confirm the diagnosis. Sometimes a protracted examination may be of service, as the movements of the cysticercus become more pronounced in cônsequence of prolonged illumination. Great caution should be exercised in asserting the death of a cysticercus. As long as the pupil permits the passage of light the cysticercus is certainly alive and will sooner or later resume its movements. In the first few years following the invention of the ophthalmoscope some cases of encapsulation of cysticerci in the vitreous, with retention of a slight amount of vision, were reported. This diagnosis was afterwards found to be erroneous. Hirschberg had an opportunity of seeing one of these cases again, and was convinced that the trouble was a congenital connective-tissue formation in the vitreous.

If closure of the pupil or the formation of cataract has taken place, a qualified diagnosis only can be made. As a rule, the diagnosis will then lie between an intra-ocular tumor and a cysticercus. Increased tension would speak for the former, diminished tension for the latter. It must be mentioned, however, that in rare cases of cysticercus an increase of tension has been found (Hirschberg, Rampoldi).

The **prognosis** in extra-ocular cysticercus is favorable; in the intra-ocular form it is always bad. Left to itself, the latter disease leads infallibly to blindness. Internal remedies—for example, tænifuges—have not the least effect on the cyst. The suggestion to puncture the cyst and thus produce the death of the entozoon was very soon abandoned as impracticable, as the parasite usually slips away from the instrument, and, even if this does not occur, it is to be feared that the dead parasite would cause more irritation than the living one. Therefore the only rational remedy is operative removal. In the extra-ocular varieties this procedure is very easy and is followed by complete recovery. In the intra-ocular forms it is often very difficult and of doubtful result.

The **extraction** of the cysticercus from the anterior chamber is simple, and the procedure comparatively old. The cornea is incised as near the parasite as possible, and the aqueous is allowed to escape. If the cyst is free in the anterior chamber, it comes out with the aqueous. If it is fixed

to the iris, it must be excised together with the portion of the iris to which it adheres. The first attempts of von Graefe at extraction of cysticercus of the vitreous were not encouraging. It was not until the meridional scleral section was brought forward by von Arlt and improved by Alfred Graefe that treatment of cysticercus in the vitreous was attended with success. It was soon found that this method could also be used for the removal of subretinal cysts, and that, in fact, the results at this stage, which previously had been considered inoperable, were better than when performed later.

The determination of the exact location of deep-seated cysticerci is an indispensable prerequisite for an operation. This may be done when there is a scotoma by simple measurement of the visual field. However, when the visual acuity is markedly reduced, the location of the cyst can be ascertained only with the ophthalmoscope. By means of this instrument the meridian in which the parasite is situated is first determined, and if necessary marked by tattooing; then its distance on this meridian from the papilla estimated in papilla-diameters. By this procedure the distance of the parasite from the edge of the cornea may readily be calculated. Owing to the location of the incision, the determination of the meridian is of much greater importance than that of the distance of the parasite from the papilla. For very exact measurements the localization ophthalmoscope of Alfred Graefe may be employed as long as the patient has central fixation. This instrument consists essentially of a perimeter carrying a movable object of fixation rotating around the aperture in the mirror of an ordinary ophthalmoscope. During the examination the affected eye must be situated at the centre of curvature of the perimeter. The patient is requested to look steadily at the object of fixation, and the latter is moved until the cysticercus becomes visible in the pupil. The meridian as well as the arc of rotation of the globe may then be noted from the instrument. After having determined the location of the proper meridian in this manner, the sclera is exposed by a liberal incision in the conjunctiva and Tenon's capsule, and, if necessary, by section of an ocular muscle. In the case of subretinal cysts the incision is cautiously made with a narrow knife, whose edge is turned towards the sclera, as it is very important that the sclera and choroid be divided without injury to the parasite. If the incision be properly made, the parasite presents itself spontaneously in the wound, and may easily be delivered intact without injury to the retina or the hyaloid membrane.

In the case of cysticercus in the vitreous the knife should be plunged into the globe up to the parasite. The point of the knife should be directed towards the sclera, and the incision made with a sawing motion. Here, too, spontaneous presentation of the cyst frequently occurs, or the parasite may escape altogether with a small quantity of liquid vitreous. In other cases traction instruments must be employed, and the delivery of the cyst, if successful, is obtained only at the cost of a considerable loss of the vitreous.

As has been indicated, the chances for success are most favorable in

subretinal cysticerci, less so in fixed cysticerci of the vitreous, and very unfavorable if the cyst be free in the vitreous. The fewer the instruments inserted into the vitreous the better the result. Above all, the stage of the disease has the greatest influence upon the success or failure of the operation. Generally we may count on maintaining the *status quo*. In some cases improvement of vision, due to readhesion of the retina, or disappearance of opacities of the vitreous, may result, and even perfect vision has been obtained in a few especially favorable cases (Alfred Graefe, Leber, Hirschberg, Stoelting). Even if the eye be entirely blind the operation is of advantage in that it relieves pain and prevents atrophy of the globe. The extraction of the cysticercus is therefore indicated in all cases in which the exact location of the parasite can be determined. Only in the last stages, where a view of the interior of the eye cannot be secured, is the idea of enucleation or exenteration to be entertained. If the eye is not irritable, although blind from the presence of a cysticercus, it may be retained without danger.

**Prophylaxis.**—Of far greater importance than any operation, be it ever so boldly and skilfully performed, is the prevention of infection by the cysticercus. As there are few diseases whose etiology is so well and definitely known, well-regulated preventive measures may do much to diminish its frequency. All plans directed against tænia solium tend indirectly to prevent cysticercus. These, however, devolve mainly upon the State. The meat markets should be carefully regulated, and the sale of any meat containing measles forbidden.[1]

The individual should take care to eat only well-cooked meat, and if, in spite of this, he should acquire a tapeworm, he must try to get rid of it as speedily as possible. It is the duty of every physician to point out to a patient afflicted with tænia its danger to himself and those about him. The most efficient measure of individual prophylaxis is scrupulous cleanliness in regard to bed and body clothing, as well as at meals. The hands should be well washed before each meal, and food or cooking utensils should not be kept in sleeping-rooms or in the neighborhood of closets.

### CYSTICERCUS OF TÆNIA MEDIOCANELLATA KÜCHENMEISTER (MEASLES OF BEEF).

The cysticercus of beef differs but little in development or structure from that of pork. It is the youthful stage of tænia mediocanellata Küchenmeister, and, like this parasite, is distinguished by the absence of hooklets. Until recently the opinion was prevalent that this form of cysticercus did not infect man at all. Deutschmann, however, observed it under the conjunctiva of a man of fifty-three. The symptoms in this case

[1] The city of Berlin furnishes an example of what can be done by preventive measures. Since the inspection of meat has been methodically carried out, ocular cysticercus has completely disappeared in that city. (Hirschberg.)

were those of cysticercus cellulosæ, the diagnosis being made after extraction of the parasite.[1] Pergens found this variety in the vitreous humor. This case was, furthermore, unique in that two cysts were present in the same eye. These cysts were characterized by the absence of hooklets, an unusual length of the scolex, and a blackish-brown pigmentation of the head.

## ECHINOCOCCUS.

**Natural History.**—The echinococcus is the youthful stage of tænia echinococcus of the dog. Its immigration and first development are similar to the corresponding stages of cysticercus cellulosæ. Its growth is much slower. It may, however, attain a considerable size. For a long time the parasite exists only as a simple immovable cyst, with a layer of delicate cells and a few muscular fibres on its inner surface. The formation of the head does not begin until the end of four full months, during which interval the cyst has attained about the size of a walnut. It also happens that still older cysts are found sterile,—a variety known as acephalocysts. The development of the head is different from that of the head of the cysticercus. On the inner surface of the primary cyst (mother cyst) a number of so-called brooding capsules develop. These are cysts in which an unlimited number of heads may develop as hollow buds. The matured heads project towards the interior of the brooding capsule, and then each appears about three-tenths of a millimetre long, with a blunt rostellum, a circle of hooklets, four sucking-disks, and a short ovate body. They are connected with the wall of the brooding capsule by a delicate pedicle.

The cyst, as well as the brooding capsules, may undergo manifold changes, producing the forms described as *E. simplex*, *E. hydatidosus*, and *E. multilocularis*. In the limited field of ophthalmology only the acephalocysts and the echinococcus hydatidosus require consideration. The former are found frequently in the orbit simply because their presence is more readily discovered in this location than in other organs. Anatomically, this parasite is characterized by the lamellar structure of the cyst-wall previously mentioned and by its clear watery contents, which are free from albumen and are rich in chloride of sodium. The echinococcus hydatidosus contains, moreover, a variable number of daughter cysts, and in some cases there are normal brood-capsules, detached heads, and free hooklets. As in cysticercus, the primary echinococcus cyst becomes surrounded by a capsule of connective tissue, which is derived from the neighboring tissues. The external manifestations of inflammation are, however, wanting.

**Etiology.**—Intimate association with the dog must be regarded as the cause of echinococcus infection. As a result of this, the disease is most prevalent among people who by their occupation have much to do with this animal and often share bed and board with it. For these reasons children

[1] The author desires to express his thanks to Professor Deutschmann for the use of the notes of this case, which have not as yet been published *in extenso*.

are most liable to contract the disease. Echinococcus is decidedly less frequent than cysticercus, and is found most frequently between the ages of ten and twenty years.

**Course.**—Two instances of echinococcus in the interior of the eye have been reported: one by Gescheidt, in 1853, and one by A. Hill Griffith, in 1896. (See "System," vol. iii.) Disregarding these cases, this parasite would appear to infest the ocular apparatus only in the orbit. Here it may occupy the most diverse positions, within or without the muscular cone, in the lacrymal gland, or in the sheath of the optic nerve. It may also occur in the neighboring cavities and thence invade the orbit, or, conversely, it may penetrate from the orbit into the cranial cavity. The limited area affected by the echinococcus is a contrast to that occupied by the cysticercus. Its most important symptoms are protrusion and dislocation of the globe and impairment of its motility by an elastic and more or less fluctuating tumor. Vision is usually considerably reduced, and may be entirely lost. The ophthalmoscope reveals choked disk or the results of this process. Severe ciliary pain is almost constantly present. The cystic fluid does not coagulate upon boiling, and the addition of nitrate of silver causes a white cheesy precipitate. Microscopic examination may demonstrate the heads or hooklets. If the cyst has penetrated the skull, pulsation may be present.

The **differential diagnosis** lies mainly between encephalocele and dermoid cyst. Both are congenital, the former showing, as a rule, communication with the cranial cavity. Exploratory puncture may be employed to distinguish echinococcus from a dermoid cyst. Echinococcus has sometimes been mistaken for rapidly developing sarcoma or tumor of the optic nerve. The prognosis is unfavorable when the disease is left to itself. The pressure on the optic nerve causes blindness. Protrusion of the globe is followed by its loss.

The **treatment** to be recommended is total extirpation of the tumor. When this, from the size of the mass or from its inaccessibility, is impracticable, it should be exposed by a free incision, its contents aspirated through a strong canula, and the collapsed mother cyst extirpated. Any remaining fragments may be removed by a curette. If desired, the cavity of the cyst may be opened at once by the first incision, its contents allowed to flow out, and its walls destroyed. The connective-tissue capsule does not have to be removed, for it belongs properly not to the parasite but to its host. The wound must, of course, be drained, to furnish a free exit for any remaining shreds. If the operation be performed in this way, with rigid aseptic precautions, there will be no suppuration, as formerly. The results of treatment are, on this account, much better now than heretofore. Indeed, the prognosis is not unfavorable when surgical interference is early, provided there has been no injury to the optic nerve fibres. A rapid improvement in visual acuity is at once noticed, but the congestion of the papilla subsides more slowly. If atrophic changes have taken place in the optic nerve

*restitutio ad integrum* is out of the question. The result is, unfortunately, sometimes rendered less favorable by impairment of the motility of the eye from formation of indentations in the orbit or injuries to the muscles of the eye or their nerves during the operation.

Prophylaxis plays as important a *rôle* in this disease as in the one previously considered. As the dog is the host of the corresponding tænia, this useful and often indispensable animal should be isolated as much as possible. Parlor dogs are a useless luxury, and ought to be banished entirely from families with children. Under no circumstances should the animal be allowed to lick the hands or face of any person.

The two parasites just considered are the only ones of pathological importance in our climate. All other entozoa which have been found in the eye or its neighborhood have been observed in isolated instances only.

Of the order of *Trematodes*, several forms have been found in an undeveloped state in the eye. Thus, for example, the *Distomum ophthalmobium Diesing* has been found in the congenital cataract of a child, whilst the *Monostomum lentis von Nordmann* has been seen in the acquired cataract of a woman. Przibilsky has recently reported the following case. A detachment of the retina was observed in the region of the macula. At its upper edge a rod-like projection was seen, which after two months had passed completely into the vitreous, while the detachment of the retina had disappeared. This object showed varying independent movements. Its lower end was bent sharply backward and flattened. Later two new processes developed from it. No operation was done.

The order of *Nematodes* has furnished several ocular parasites. In severe cases of trichinosis the extrinsic muscles of the eye may become involved. The symptoms are œdema of the lids, pain, and impaired mobility, as well as exophthalmos (Kittel).

*Filaria loa Guyot* has been known for almost a century. This worm is usually from thirty to forty millimetres long, has an unarmed mouth, and looks not unlike a fine violin-string. It is found under the conjunctiva of the negroes of the west coast of Africa from the Gold Coast to Angola (5° north to 10° south latitude). The free motility of the worm is very striking. It wanders in all directions under the conjunctiva, and not infrequently from one eye to the other, across the bridge of the nose. It may also appear under the skin in other parts of the body. Warmth tends to bring it to the surface. Under the influence of cold it disappears without a trace in the deeper tissues. In the latter situation it gives rise to no annoyance. It is only when it appears under the conjunctiva that the eye is made to water and to become injected. The life of the parasite extends over several years. It finally disappears spontaneously without any unpleasant after-effects.

We know but little of the natural history of the filaria loa. The filaria diurna that by day is found in the same places in great numbers in the

blood of the negroes is supposed to be the embryo of the filaria loa. It is believed that these embryos are sucked up with the blood of the negroes by the mosquitoes, that they undergo further development in these intermediate hosts, and are then deposited in the water with their eggs. Water contaminated in this way may cause reinfection of man. Infection by the loa would have little significance in our latitudes were it not that recently white men who have visited the part of Africa above alluded to have contracted the disease (*vide* Robertson, Saemisch, Barrel). The present increased activity in African colonization will doubtless make the loa better known in Europe. Its acclimatization is, however, not to be feared, since the conditions favoring its propagation are present only in the circumscribed area of its normal habitat. The removal of the parasite is easily accomplished by incising the connective tissue. Care must, nevertheless, be observed to hold the worm fast by digital pressure or with a forceps, as it will otherwise quickly escape. It is difficult to avoid injuring so frail a creature during its extraction. This, however, is attended by no untoward results. As a prophylactic measure, sterilization of drinking water is most urgently to be recommended.

*Filaria inermis Grassi* is a parasite that is frequently found in the horse and ass. Up to the present time it has been observed in man in only three instances, in Italy. In Addario's case it was located in a tumor about the size of a pea in the conjunctiva. In the case reported by Pace it was situated in the subcutaneous tissue of the eyelid. No details of the third case are given. The worm may attain a length of nine centimetres and more.

*Filaria medinensis Gurel* occurs occasionally under the skin in the neighborhood of the eye, but is said never to be found under the conjunctiva. Although filiform worms are often met with in the anterior chamber in the lower animals, there are very few authentic cases that have been seen in man; and in these it was a question of unripe—that is, zoologically indeterminate—worms. A case of this kind was observed in Belgium in a two-and-a-half-year-old negro girl, who had come from the Congo region. The case was described by Gauthier, Henri Coppez, Lacompte, and van Duyse. The worm appeared to be from two to three centimetres long, and was in continual motion. The eye showed no signs of irritation, and the media were clear. After a time the worm died spontaneously, and lay coiled up in the fornix of the anterior chamber. Iritis then set in. The worm was easily removed by an incision, and the eye became better. The worm proved to be an unripe loa. Drake-Brockman observed a similar case in a young woman from Madras. Barkan's case is doubtful. The presence of undeveloped filiform worms in cataractous lenses seems at one time to have been established by von Nordmann and Gescheidt, though similar cases have not been observed since then. Kuhnt once saw, in the partially cataractous lens of a young recruit twenty-one years old, a thread-like object curled up in a spiral that he was inclined to regard as a nematode worm,

although he was unable to positively identify any subjective movements in it. Hennicke attributed a unilateral cataract in a fourteen-year-old boy to entozoa, for no further reason than that the boy had previously suffered from oxyuris. There was no anatomical confirmation of either of these cases.

Of considerably greater diagnostic interest are the cases of filariæ in the vitreous. The cases of Quadri, Fano, Mauthner, and Schoeler have been thoroughly discredited by Kuhnt, who declares them to have been remains of the hyaloid artery. The only anatomically authenticated and therefore indubitable case of thread-worm in the vitreous was reported by Kuhnt himself. A healthy man of thirty-one developed a central chorio-retinitis with slight retinal detachment. After a few months an opacity became visible in the vitreous in front of the macula, while the detachment of the retina at the same time receded. The opacity gradually increased to the diameter of the papilla, and showed traces of independent movement at the end of over six months. The patient suffered so intensely from photopsia and pain that in spite of the dubious prognosis he accepted operation. The opacity was removed through a meridional scleral incision, and was found to contain a coiled worm 0.38 millimetre in length. This was pronounced by Leuckart to be the youthful form of the filaria or of the strongylus. In 1891 Eversbusch demonstrated, at Heidelberg, a living thread-worm in the vitreous. The promised exact report on this case has not been published. These isolated and in part very doubtful cases do not suffice to enable us to furnish a comprehensive description of the symptoms caused by these rare guests in the vitreous. A comparison of Kuhnt's case with that of Przibilsky shows that the occurrence of a limited detachment of the retina, receding after the formation of an opacity of the vitreous, is of some diagnostic importance. At all events, the diagnosis of a living thread-worm in the vitreous is one of the most difficult in the field of ophthalmoscopy, because any filiform formation in this humor—as, for instance, a persistent hyaloid artery—has a high degree of mobility, while persistent oscillatory vibrations may be excited in it by the movements of the eye. If such a structure be so situated that it is seen foreshortened, these movements may easily be mistaken for intrinsic changes of form.

# SIMULATED BLINDNESS.

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INTELLIGENT and well-informed malingerers are well aware of the difficulties of diagnosis in those cases in which loss or impairment of vision is unexplained by any evident anatomical change, the causal lesion being so slight or so situated that the methods of investigation at our disposal do not enable us to detect it. Feigned amaurosis and amblyopia, moreover, occupy the front rank, as to frequency, among the ocular affections which impostors feign, exaggerate, provoke, or conceal.

The motives which lead to malingering are most diverse. It may be a question, perhaps, of evading certain obligations or social duties, such as military service; or of exaggerating the consequences of an injury, in order to receive the highest damages from an employer or from an insurance company. Some individuals, without means of support or desire for work, feign complete blindness in order to obtain a pension or admittance into a charitable institution. With the supposed object of avoiding banishment, and of being allowed to remain near his family, the prisoner, whose case Beauvais[1] recently presented to the Society of Legal Medicine of France, feigned for a period of six months a double amblyopia, which he attributed to effect of starlight. At other times, hysterical subjects are seen who, as a result of vexation, through revenge, fancy, etc., declare themselves to have become suddenly and totally blind. Finally, children, to whom school life is a burden, complain of fictitious ocular troubles, in the hope of returning home, or even with the object of making a journey to consult a medical specialist (Pansier's case[2]). Some persist in their assumed *rôle* with a

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[1] Simulation d'amblyopie double attribuée à la lumière des éclairs. Annales d'hygiène publique et de médecine légale, 1896, p. 434.

[2] L'Amaurose hystérique; dédoublement de la vision consciente et de la vision polygonale. Annales d'Oculistique, 1897, pp. 402–407.

tenacity which is only overcome by preparations for surgical interference (Hirschler's case [1]).

From another point of view malingerers can be classed in two categories: First, the artless and the ignorant patients, who often have nothing wrong, and whose imposture is easy to expose by the simplest methods; second, the intelligent, the well-informed, and the tenacious subjects, who have rehearsed their parts for a long time, and only exaggerate a real visual defect. The latter cases demand, on the part of the physician, a large fund of special knowledge, experience in making investigations of this kind, a great deal of apparatus, and much sagacity and patience to induce such malingerers to admit of trickery; and even then, in spite of oft-repeated examinations, it is sometimes found impossible to determine with exactness the real degree of diminution of vision.

We are led to suspect malingering when the functional disturbances do not correspond with the objective signs; for example, if the patient denies experiencing any sensations of light, although changes of illumination occasion pupillary movements; when the results of examination of direct vision of the field of vision, of color perception, and of the light-sense, at different distances and at different times, are contradictory; and when the patient's circumstances and behavior suggest that he has a tendency or an interest to feign or to exaggerate an imperfection of vision. Suspicion, however, does not suffice. The deception must be demonstrated by a series of proceedings, some of which merely show the bad faith of the patient, while others allow the degree of visual acuteness which he enjoys to be established with sufficient exactness.

It is certain that neither means of taking these people unawares, nor sufficient proper apparatus for unveiling the impostures, is lacking; their multiplicity, however, is evidence that no one of them is thoroughly satisfactory. Moreover, this question of methods and apparatus remains in a certain sense always open, because in proportion to the frequency of their employment the most ingeniously contrived procedures lose their efficacy, the various methods of action finally becoming known to intelligent malingerers, especially to those having some idea of optics. It will always, therefore, be useful to introduce modifications of methods already in use, and to be familiar with unpublished ones, in order to bewilder trained malingerers.

Frölich [2] has made a study of the most interesting of the numerous recent procedures for detecting malingering. Only the most important will be discussed here, from among which the physician will still have to select those which appear to him best suited to the particular case upon which he is called to give an opinion.

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[1] Simulirte Blindheit des rechten Auges. Wiener medizinische Wochenschrift, 1861, S. 827.

[2] Des procédés modernes pour reconnaître la simulation de la cécité ou de la faiblesse visuelle. Revue médicale de la Suisse Romande, 1891, p. 721.

The patient most frequently complains of complete blindness or defective vision on one side (*amaurose ou amblyopie unilatérale simulée*). Exceptionally he tries to prove that he is completely blind, or that the sight of the two eyes has become defective (*amaurose ou amblyopie bilatérale simulée*).

## I.—SIMULATED BLINDNESS OF ONE EYE.

After having assured himself by an exhaustive clinical examination that the visual symptoms are not accounted for by a lesion situated either in the deeper membranes of the eye or in the central visual nervous apparatus, the physician should investigate first of all (1) *the condition of the pupil* and (2) *the direction of the visual axis.* Von Welz's test will accomplish this examination satisfactorily.

### A. OBJECTIVE METHODS.

The testimony of objective observation is the most incontestable. Unfortunately, this method of examination cannot teach anything concerning the degree of visual acuity which the malingerer possesses.

#### 1. CONDITION OF THE PUPIL.

The reaction of the iris to light-stimulus is extremely definite, and constitutes a valuable sign which allows the physician to ascertain objectively whether the retina is sensitive to light. Before investigating pupillary motility, however, it is well to make sure of the absence of miosis, synechiæ, or inflammation, as well as of psychical or sensory stimulation. The patient must be placed in an apartment which receives light through a window in such a way that the eyes are equally illuminated. Strong and feeble illumination should be successively employed. If the right eye is blind and the healthy left one is covered,—but in such a way that it can be observed, —there will be complete immobility, not only of the iris of the right eye, when it is first covered by the hand and then exposed to diffused sunlight while looking at a distant object, but also of the iris of the left eye; the sphincter of which would be sympathetically contracted if the retina of the right eye retained sensibility to light.

The patient being next placed in a dark room, the amaurotic right eye, for example, is to be covered. The physician, while continuing to observe it, should throw a pencil of light on the healthy left eye, varying the intensity of the illumination, or make the patient fix with this eye upon an object that is placed very near and towards the nose (accommodation and convergence). In each instance the pupil of the right eye will contract if the iris is not under the influence of a mydriatic. The method recommended by Arlt for the purpose of ascertaining if the retina is sensitive to light consists in illuminating the macular region of the retina for a brief period of time, when, if light-perception be preserved, blinking and some lacrymation will be observed.

An examination of the pupil becomes a delicate and a doubtful matter if the eye, amblyopic only, still possesses quantitative perception of light,

for in this case any opinion may be erroneous, to the detriment of the patient. No conclusion, therefore, except that which is obtained from a number of tests, controlling one by another, should be given. The same applies when a real or an artificial mydriasis exists. An eye with a dilated pupil is far from always being an amaurotic one. It may be only amblyopic, or it may possess a normal visual acuity, although presenting, in the latter case, functional disturbances resulting from dazzling and loss of accommodation. The following plan is the best method of making a differential diagnosis. The patient is placed in a dark room. The degree of contraction of the sphincter of the healthy left iris when light is focussed upon the eye is to be observed. The eye is then to be screened; this done, the light is thrown by means of a lens on the right eye with its dilated pupil. If the retina of the latter eye is insensitive, the pupil of the left one will not vary: if the right eye is amblyopic only, the left pupil contracts slowly and feebly; if its visual acuity is normal, the contraction of the left pupil is as pronounced as if luminous rays are thrown directly upon the left eye.

Mydriasis is often induced with fraudulent intent. It behooves the physician not to be misled by it. Cases are common in which the imposture is a very shallow one, soon resulting in the confusion of the too simple malingerer. In such cases the pupil is dilated *ad maximum;* the iris is almost imperceptible, so much of it being effaced. Sometimes there remains a slight conjunctivitis of special type that is due to the prolonged use of the mydriatic. When, however, the malingerer is well informed, he makes use of a minute dose of the drug, and ceases the instillations some time before the examination, so as to obtain only a medium dilatation. In such a case doubt is permissible, it being necessary to make such arrangements that the malingerer cannot again have recourse to the mydriatic. In artificial mydriasis, stimulation of both retinæ produces no contraction of the pupil.

To recapitulate: the fact that the right pupil remains motionless when exposed to a bright light, while it contracts under the influence of convergence and accommodation, points with great probability to the existence of a *unilateral amaurosis.*

In the case of amblyopia, examination of the iris and pupil will furnish much less conclusive information than when it is a question of the simulation of *unilateral amaurosis.* An *amblyopic* eye still possesses, in fact, *quantitative* perception of light. Unless there is simulation, or indeed a true or artificial mydriasis, a more or less marked *sluggishness* of the movements of the iris compared with those of the healthy eye should be demonstrated. It will be easily understood that there may exist only minimum differences, the interpretation of which is difficult and therefore doubtful. It is always necessary to remember, in this examination of the light reflex, that the iris-reaction may be lacking in an eye that is sensitive to light, in the same way as in cases in which the iris reacts normally, although the retina is no longer sensitive to impressions by a luminous agent.

In hysteria the iris generally responds to light, but it may be contracted or dilated and fixed.

Westphal[1] says miotic pupils occur either independently of an attack of hysteria or as the result of a slight one. He has also seen the paradoxical iris-reaction, and a well-marked case of hippus. The dilated and fixed pupil occurs usually during convulsive attacks (Karplus[2]), which leave no doubt as to the character of the case, and is not likely to be met with in persistent hysterical simulation of blindness. That it may be seen, however, even in monocular simulation, is shown by the following case:[3]

A girl, sixteen years of age, with a decidedly hysterical history, asserted that there was no vision in the right eye, except a vague perception of light. There was also violent clonic spasm of the orbicularis muscle and of the muscles of the face. The pupil was widely dilated and fixed. The left eye was normal. With a + S. 3 D. lens placed before the right eye and a + S. 18 D. lens situated before the left one, she read two and one-half Snellen type at fourteen inches' distance,—of course with the "blind" eye. As the muscular spasm was said to have been relieved on a former occasion by the application of a Charcot magnet, an imitation of this magnet, made of wood with metal tips, was used, with the effect of relieving the muscular spasm and at once restoring the pupil to its normal size and the iris to perfect reaction. This mydriasis, promptly cured by mental impression, must have been due to an excitation of the pupil-dilating centre in the medulla, which is known to be influenced by the emotions.

If an eye is amaurotic, or is extremely amblyopic, it has a manifest tendency to deviate while its normal fellow is directed towards any point looked at. When there is no evident deviation of one eye, it is sufficient, in order to ascertain if the patient fixes exactly with both eyes, to alternately cover the right and the left eye while making him look at the flame of a candle placed two or three metres distant. If binocular fixation exists, neither eye will move when the other is covered. The diagnosis of the integrity of binocular single vision will be completed by the use of a prism (diplopia and deviation or displacement of the eye) and by different tests, such as the Holmes stereoscope and the plates of Dahlfeld or of Kroll, etc.

In accordance with Schmidt-Rimpler and Pickert, the writer finds that Hering's experiment, recommended by Schweigger, tends to errors of observation, even with patients with normal vision, this being much more so, therefore, with but slightly intelligent or inattentive subjects. Of less value also is the phenomena of *lustre* or *stereoscopic radiancy* (Dove), which is obtained by the fusion of two pictures, one of which is drawn in black on a white ground, and the other in white on a black ground.

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[1] Berliner klinische Wochenschrift, 1897, 47 und 48.

[2] Wiener klinische Wochenschrift, 1896, Nr. 52.

[3] Harlan, Transactions of the American Ophthalmological Society, vol. iii. p. 649.

The patient may have single vision with the two eyes open when one eye is blind, or when he suppresses the impression that is received by it (monocular vision). Monocular vision very often accompanies strabismus, an affection which it is very difficult, not to say impossible, to feign during prolonged examinations.

Paralytic strabismus of recent date frequently occurs without impairment of the visual acuity of the affected eye; but the contrary also holds good. As for non-alternating strabismus, this is most frequently associated with an anomaly of refraction and with a diminution of visual acuity, which may be either congenital or acquired. This form of amblyopia continues to progress in proportion to the duration of the strabismus, and finally scarcely allows the patient the ability to see to count fingers at any distance.

In these cases all possible information should be obtained, and minute examinations should be made in such a way as to base an opinion on repeated trials, the value and results of which cannot be open to suspicion. As a matter of fact, the physician may be entirely baffled, for the subject affected with functional strabismus possesses the faculty of ignoring the image received by the right eye, which he has declared blind. As a result of this psychical exclusion, the vision of such a patient is therefore monocular,—that is to say, such a subject does not habitually enjoy binocular vision.[1]

It should be remembered that stereoscopic fusion is not only a physiologic phenomenon but also a central psychic act (Janet, Bernheim, Meyer, etc.), and that with certain patients with strabismus there exists a kind of dissociation of binocular vision (Schweigger, Greeff, Simon, etc.), which is not equally perfect in the use of the stereoscope, in the act of reading, etc., just as cases of monocular amaurosis of a hysterical nature with retention of binocular vision (Charcot, Panas, Westphal, Dor, Jr.[2]) which are sometimes dissociated may be observed (Antonelli[3]).

The test known under the name of the von Welz method consists in placing a prism of from ten to twenty degrees, with its base outwards, in front of the eye that is supposed to be blind, and endeavoring to make the patient read. In such a condition, to obtain single vision, the right eye will deviate inward and recover itself the moment the prism is removed,—movements which the malingerer will not make his eye undergo should it not really see.[4]

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[1] Sachs, Ueber das Sehen der Schielenden. Archiv für Ophthalmologie, Band xliii.

[2] Une observation de persistance de la vision binoculaire dans un cas d'amblyopie monoculaire hystérique. Revue générale d'Ophtalmologie, 1897, p. 51.

[3] La dissociation de la vision binoculaire chez quelques strabiques et quelques hystériques, à propos d'un cas d'amaurose monoculaire hystérique. Archives d'Ophtalmologie, 1897, p. 218.

[4] Welz, Ueber Entdeckung simulirter Amaurose und Amblyopien. Congrès interne d'Ophtalmologie de Paris, 1867.

## B. SUBJECTIVE METHODS.

Besides the evidences of objective observation, there remains a recourse to methods which depend upon catching the patient unawares (properly called "de surprise"), all of these plans being based upon the principles of optics or of physiology. A great number of these methods rest upon the properties of plane mirrors, of concave mirrors, of prisms, etc. In others (the methods of Javal-Cuignet, Snellen, etc.) the patient is made to try to read with the supposed blind *right* eye, for instance, some letters which the healthy *left* eye cannot distinguish by the interposition of a screen between the presumably good eye and the letters. The eye is, *par excellence*, the organ of illusions, and the methods of deceiving it are numerous. As the writer has previously said, among these tests some tend to establish only the dishonesty of the patient, while others allow a sufficiently accurate determination of the visual acuity of the eye examined.

*It is taken for granted that, in all these experiments, the physician takes care that the patient does not succeed, under any pretext, in closing either eye, even momentarily.*

### 1. JAVAL-CUIGNET'S METHOD AND ITS MODIFICATIONS.

The description of the subjective methods are here begun with the plan of Javal-Cuignet, it being one of the simplest and one of the oldest. The test can also be employed for the verification of the existence of binocular vision.

*Javal-Cuignet's Method.*[1]—After the physician has tried this method on himself, he is to hold fixedly, some thirty-five centimetres from the eyes to be examined, a sheet of paper on which are traced dots, figures, and printed letters. A pencil or a finger is to be interposed at an equal distance from the nose and the paper in the median line, in such a way as to render some of the letters, figures, or dots invisible in each of the visual fields. If the patient reads these letters or the whole line accurately, the deception is detected. By taking into consideration the size of the letters which have been recognized, an approximate measure of the visual acuity can also be obtained.

Apparently very simple and not requiring any costly apparatus, the Javal-Cuignet method presents some difficulties in its application. It requires absolute immobility of the head and a printed page; the physician also must be able to fix in his own mind the details of the test and to familiarize himself with it in order to interpret with rapidity and certainty the answers of the patient. So as to lead the patient more surely into error, it is advantageous to make him perform lateral movements of the eyes by moving the finger, a pencil, or even a sheet of paper. The

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[1] Cuignet, Note sur un moyen de constatation de l'amblyopie et de l'amaurose d'un œil. Recueil de mémoires de medécine, de chirurgie et de pharmacie militaires, xxiv., 320, et xxix., p. 257.

paper should, by preference, be transparent, for by standing behind, with the eyes above its upper margin, it can be very easily noticed which is the point or letter that should disappear in accordance with any lateral movements that may be induced.

The modifications added to this method, successively by Martin[1] and Barthelemy,[2] allow the experiment to be made with more precision, and performed for the establishment not only of the existence of amaurosis, but also for the measurement of visual acuity.

*Martin's Apparatus.*—This has been constructed with a view to applying the Javal method. It consists of a box thirty-five centimetres long by twenty centimetres broad. In front are two openings which serve for eye-pieces, and the test letters are arranged on the posterior surface. Fifteen centimetres in front of the latter, and in the median line, a rod one centimetre in diameter, which can be raised or lowered according to necessity, is placed.

Facing the window, the examiner holds the apparatus between his hands, the posterior wall of the box being placed against his chest. He is then to make the patient look through the anterior opening, the rod having been previously raised. If the patient reads all the letters, the fraud is discovered and the visual acuity is measured. Should the malingerer declare, on the contrary, that he can only read certain letters, it is merely necessary to make sure that they are in reality hidden by the rod from the eye that is claimed to be blind.

*Barthelemy's Apparatus.*—This contrivance has the advantage of serving also as a stereoscope and a pseudoscope. It consists of a large square ruler fifty centimetres long, graduated in centimetres, and supported at its middle by a pedestal or by a handle. At one of its extremities an iron plate pierced by two holes for the eyes, and furnished with hooks that permit the adjustment of prisms (stereoscope) or correcting glasses, is fixed. On the ruler, two travellers slide. One of these carries a wooden frame serving for a screen, and the other holds a test-card. A system of two mirrors that are movable around an axis that is fitted in one of the travellers can be adapted to the apparatus, allowing the physician to reproduce the tests of the Fles's box by fixing the cards or test-letters in a position that is external to the apertures of the screen which is intended for the eyes of the patient.

*Driver's*[3] *Method.*—Driver interposes a vertical ruler, four centimetres broad, at a definite distance between the eyes of the patient and two of Snellen's test-types in such a way that the ruler acts as a screen hiding the right test-type from the left eye, and the left test-type from the

[1] Note sur un moyen de reconnaître et de mesurer l'amblyopie unilatérale. Recueil de médecine et de chirurgie militaire, t. xxxiv., 1878, p. 307.

[2] L'examen de la vision, etc., 1889, p. 200.

[3] Beitrag zur Entdeckung simulirter einseitiger Amaurose, Berliner klinische Wochenschrift, 1872, S. 143.

right eye. If the patient succeeds in reading the letters on the two scales, he discloses his fraud, and at the same time indicates the degree of vision of the eye which he claims is defective.

## 2. TESTS BY PSEUDOSCOPIC APPARATUS.

Many of the methods here employed are applications of the properties of the plane mirror, their object being to induce the malingerer to read letters which he thinks he is seeing with the healthy eye with the eye that he declares to be defective.

In 1860, Fles[1] contrived an ingenious instrument intended to make a supposed amaurotic eye see an image which the patient imagines he is seeing with the good eye. It consists of a rectangular box in which two mirrors of a definite size and orientation are placed vertically under an inclination of one hundred and twenty degrees.

The small dimensions of the apparatus, causing prolonged efforts of accommodation before the images are found, and the images being formed so near to one another that they have a tendency to blend, produces a lack of precision in the answers of the patient. Consequently, with the object of rendering the plan more practical, Fles's box has undergone modifications.

Baroffio inclines the mirrors at one hundred and twenty-five degrees; Binnendijk and Armaignac[2] make them movable on a hinge in such a way as to vary the angle which they form, and to obtain such relations of the images that, without closing one of the eyes, it is impossible to know which is the image perceived by the right eye and which is seen by the left eye.

In *Monoyer's*[3] *double apparatus* the two mirrors are placed on a line parallel to the plane which joins the eyes of the person examined. This arrangement is preferable to that of Mareschal's[4] pseudoscope, which has but one mirror. In addition, the transverse dimension of the box is such that the patient can see the images of his own eyes at the same time as the images of the objects or test-drawings, which is not the case with the apparatus of Fles, Armaignac, and Monoyer, owing to the small size of the mirrors and to their orientation. Moreover, the screens inserted in the floor of the Monoyer box allow the mirrors to be shut off and inclined as in the Fles apparatus. Marini[5] has adapted prisms to the eye-pieces in such a way as to perform the test of A. von Graefe, and to convert the Fles apparatus into a stereoscope. Other additions, among which is an angular screen provided with a median window, allow also Cuignet's experiment and other hemioscopic tests to be made. In the apparatus of Mareschal

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[1] Nederlandsch Tydschrift voor Geneeskunde, 1860, p. 309.

[2] Traité élémentaire d'ophtalmoscopie, d'optométrie et de réfraction oculaire, 1878, p. 451.

[3] Wundt et Monoyer, Physique médicale, 1884, p. 323.

[4] Note sur une modification à la boite de Fles. Recueil de mémoires de médecine, de chirurgie et de pharmacie militaires, 1879, p. 437.

[5] Astegiano, Giornale medico del regio esercito e della regina marina, 1889, p. 242.

there is but one mirror, this being small enough to prevent the eye from seeing the sign which is placed on the same side. The box is sufficiently large to prevent any effort of accommodation and convergence (strabismus).

Astegiano[1] has attempted to convert the Fles box into an apparatus permitting the visual acuity of an eye that has been declared amblyopic to be exactly measured.

A graduated scale of reversed letters, or of symbols for the illiterate, is substituted for the test-cards (pictures of playing-cards). The upper wall of ground glass is discarded, because it diminishes the illumination to too great an extent. In its place, a mirror which is mounted on hinges and acts like that of a stereoscope is made to reflect daylight into the interior of the box. Metallic mirrors are used in preference to ordinary ones, in order to obtain clear images and to avoid multiplicity. Finally, the eye-pieces are made movable in such a way as to be capable of adjustment to the variable intervals between the eyes of different patients.

The Fles apparatus and the different improvements that it has undergone merely allow the physician, in case the experiment is successful, to assert that the person examined is not completely blind in one eye, not permitting any approximate information as to the visual power of the eye which has been declared defective. In the latter case the patient compares the two halves of the scale which is shown him, and, by a difference of the clearness of the image, takes note of the letters that are seen by the amblyopic eye, and so reads from this half of the scale merely what he chooses.

In addition, one cause of error in the measurement of visual acuity by these procedures results from a weakening of the intensity of light by the reflection of the images in the mirror, and by a dispersion of light at the point of incidence; the rule being that the more perfectly the mirror is polished the greater is the distance at which reading by reflection is rendered possible.

The optoscope of Bertin-Sans[2] consists of a box that is sixteen centimetres long, eighteen centimetres broad, and four centimetres deep. All the sides are opaque except the anterior wall, which presents two openings fitted with transparent glass, forming eye-pieces. At the side of these are two other round openings furnished with ground glass. Unknown to the malingerer, the finger can easily cover the latter, by means of a projection of wood which conceals them. In the interior of the box, on the opposite posterior wall, are two plane mirrors, the junction of which forms a projecting angle, and whose vertical planes cut at a right angle through its middle makes a horizontal line drawn from the centre of the ground glasses to an imaginary point situated twenty-five centimetres from the two eye-pieces,—that is to say, at the distance of distinct vision. As a

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[1] Giornale medico del regio esercito e della regina marina, 1889, p. 241.

[2] Nouvel optoscope pour déjouer la simulation de l'amblyopie et de la cécité monoculaires. Annales d'hygiène et de médecine légale, 1885, p. 340.

result of this arrangement, the image of each of the ground and transparent glasses is seen by each of the eyes in the shape of a small luminous circle. When the images become superposed, the luminous circle becomes single. If one of the ground glasses be blocked from use, the circle will remain single but less brilliant, and it becomes impossible to distinguish whether it is the image of the right or of the left aperture which has disappeared. The remainder of the test is easily understood.

The tests with the plane mirror, according to Herter,[1] Delay,[2] and Rosanow,[3] depend on the following principle. If both eyes are alternately illuminated somewhat rapidly and repeatedly with an ophthalmoscopic mirror, the patient cannot tell into which eye the rays are directed, because of the intercrossing of the fibres of the optic nerve at the chiasm. The writer has many times tried this experiment with an ordinary ophthalmoscopic mirror, and can avow that it has frequently given him contradictory results. An intelligent malingerer will be able to recognize in which of his eyes the image is formed, from the shape of the source of light, the dimensions of the mirror, its orientation, and the movements of the hand manipulating it. In order to remedy these defects Delay has suggested a modification which appears to the writer to be advantageous in the investigation of feigned blindness.

"It will be necessary for this experiment," says Delay, "to make use of a mirror of small dimensions (two centimetres square, for example) placed in front of a source of light of sufficient intensity and size. We obtain thus a very evenly lighted surface, the appearance of which can be in no way changed by the movements which the examiner makes to direct the reflected rays to the right or to the left. The mirror, whatever its position, will appear thus as a single luminous point. Moreover, the experimenter can place himself behind a screen, which shuts off everything except the glass of the mirror, and work far enough away, so that the movements which he impresses on it to vary the direction of the rays, may be sufficiently insignificant to be unobserved by the person who is examined.

"Some signs or test-type letters traced (reversed) on a sheet of glass, or cut out in black paper and pasted on transparent paper, are brought before the lamp, which is placed behind the patient. In illuminating alternately and rapidly, and by making the patient name the reflected signs, the physician will thus determine the visual acuity of the eye that has been declared to be amblyopic (the distance at which the sign is recognized being represented by the distance of the mirror from the patient examined, and of the test-signs from the mirror)."

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[1] Zur Entlarvung der Simulation einseitiger Amaurose und Amblyopie. Klinische Monatsblätter für Augenheilkunde, 1878, S. 385.

[2] Des principaux moyens de reconnaître la simulation de l'amaurose unilatérale. Thèse de Montpellier, 1887.

[3] De la découverte de l'amaurose et de l'amblyopie monoculaires. Vestnik oftalmologii, 1889, March and April.

The method of Coronat[1] for the detection of simulated unilateral amaurosis depends on the properties of concave mirrors. It has the advantage of being inexpensive, since it can be conducted with a concave ophthalmoscopic mirror and a card upon which some wafers are pasted; it also permits the easy watching of the patient's eyes.

To a vertical standard a concave mirror is attached, in front of which at a convenient distance is arranged another standard that is perforated with an aperture which encases the head of the person to be examined. Below this aperture is a rotating disk, which brings its colored segments one after the other into the right or the left of two horizontal apertures in a black plate which covers it. The axis of the mirror, inclined at a certain degree to the horizon, passes through a point that is situated between the eyes of the patient and the rotating disk, just as the plane of vertical symmetry of the apparatus corresponds to the centre of the mirror and to the middle of the distance of the two eyes. According to these arrangements, the color of the right aperture, for example, projected to the centre of the mirror, will be formed at the left and can be seen only by the left eye of the patient, and *vice versâ*, which he is unaware of unless he closes alternately the two eyes.

A certain number of the instruments described as modifications of the Fles box differ from it merely in the absence of the mirror. The transposition of the images is obtained by means of one or two screens (the boxes of Bertelé,[2] d'André[3]).

Prato's hemioscope is constructed of two crossed cylinders, from fifteen to twenty centimetres long, that are closed at the objective end by a transparent diaphragm, on which an object that is different on the two sides is drawn.

Melskens[4] presented at the International Medical Congress of Copenhagen (1884) an apparatus in which a movable plate enables the physician, by means of a simple mechanism, to cover sometimes the two lateral openings, and sometimes the middle opening in the posterior wall, and thus obtain at will both crossed and direct vision.

The different instruments which the writer has passed in review do not give sufficiently precise information as to the degree of visual acuity of the eye that has been declared to be defective. This is not true of the following contrivances.

*Chauvel's Method.*—The Chauvel box is rectangular, thirty-three centi-

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[1] Procédé destiné à découvrir la simulation de l'amaurose unilatérale. La Province médicale, 1893, October 12.

[2] Note sur une modification à la boîte de Fles. Recueil de mémoires de médecine, de chirurgie, et de pharmacie militaires, 1880, p. 297.

[3] Modification pratique apportée à la boîte de Fles. Recueil de mémoires de médecine, de chirurgie, et de pharmacie militaires, 1882, p. 4.

[4] Appareil pour constater la simulation d'amaurose d'un œil. Congrès international périodique des sciences médicales, Copenhague, 1884.

metres long and about twenty centimetres broad. Its posterior wall consists of a sheet of glass, divided into two parts like a stereoscopic card, on which are printed letters of dimensions that have been calculated in such a way as to measure the visual acuity at a distance of thirty-three centimetres, which is that ordinarily used for reading. The first line answers to $V = \frac{1}{10}$ and the last to $V = \frac{1}{2}$, for example. The anterior wall of the box carries two hoods projecting rather more than a centimetre's distance, and sufficiently separated to allow the nose to be placed between them. The physician, placed at one side, sees that the eyes remain exposed during the examination. A transposition of the letters is effected by two thin pieces of wood that are attached perpendicularly one over the other, by one of their borders which forms the axis around which they move. One of these pieces, pierced with two lateral holes, gives opportunity for direct vision; the second, which has only a median aperture, is arranged for crossed vision. The openings are provided with weak prisms that facilitate the dissociation of the images, it being sufficient to raise or lower a lever, that is hidden from the person examined, to obtain at will one or the other image by a displacement of the wooden plates. If the patient reads the complete lines he possesses equal visual acuity in the two eyes, the degree of which is indicated by the finest letters which have been read. By taking care in crossing the images, there is opportunity of taking the suspicious malingerer by surprise, who would read only half the line. If necessity arises, the test can be varied several times by crossing and uncrossing the images.[1]

Bonalumi has made some modifications in the details of the box.

With the idea that the sight of seven lines of letters printed progressively smaller in size, on a glass plate, may excite the suspicion of the patient, and lead him to exaggerate his amblyopia, René[2] has proposed to replace the single and fixed glass plate of the Chauvel apparatus by separate plates, reproducing only one line of letters, called "antique,"—that is, letters in which all the limbs have the same thickness,—instead of ordinary letters that have broad down strokes and thin up strokes. Other modifications depend on the arrangement of the eye-pieces, the fixation of the apparatus, etc.

### 3. TESTS BY UNCOLORED GLASSES.

*Spherical Lenses.*—Trial with plane glasses, or with very weak lenses (0.25 D.) (Arlt), with which the patient pretends that his sight is considerably improved or diminished, constitutes a test for malingering, but has not the value of the following experiments.

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[1] Diagnostic de l'amblyopie unilatérale simulée: appareil de Fles modifié. Archives de médecine et de pharmacie militaires, 1885, p. 129.

[2] René, Optométrie. Modification à la boîte de Chauvel. Système de Fles, etc., pour la mesure de l'acuité visuelle d'un œil séparément. Revue médicale de l'est, 1891, p. 228.

It is taken for granted, in these various tests, that objective examination (skiascopy, ophthalmoscopy, keratoscopy) has previously shown that the patient is practically emmetropic.

He is then placed before Snellen's test-types or some other typographical scale. A trial frame containing a plane glass or very weak lens for the eye said to be amaurotic or amblyopic, and a very strong convex or concave lens, from ten to twenty diopter's strength, for the other eye, is placed before his eyes (Harlan,[1] Dujardin,[2] Michel,[3] Vossius, etc.). He is at once urged to read with both eyes open. If he succeeds, it is with the eye that he has declared to be defective, and his answers indicate approximately the degree of his visual acuity. This may be determined more exactly if, as Wicherchiewicz[4] recommends, after having ascertained by objective methods the presence of a refractive error, the correcting lenses be placed before the suspected eye.

Silex, before putting a strong convex lens in front of the sound eye, places a series of plane or very weak concave lenses without regular order in front of the same eye.

The normal eye can by means of a six-degree convex lens be rendered artificially myopic, thus making it unable to read small letters farther away than about seventeen centimetres. The patient is asked to read at a short distance and the distance of the test-types is gradually increased appreciably beyond seventeen centimetres. If the ability for reading continues to be possible from seventeen centimetres on, it can only be done with the eye that has been declared to be defective.

In this experiment, as in the preceding, it is easy to convict the patient of his trickery at once by asking him to continue reading after the suspected eye has been closed.

On the same principle, the instillation of a few drops of atropine or of eserine into the conjunctival sac of the healthy eye has been recommended (Baroffio[5]). If, for instance, after some drops of distilled water have been instilled in the eye that has been declared defective and a few drops of eserine have been placed in the normal eye, it is found that the patient can read at the distance, it is very good proof that he sees the letters with the eye that was declared to be amaurotic or amblyopic. It seems to the writer preferable to make use of a solution of hydrochlorate of pilocarpine instead of one of eserine, because the former drug has not the disadvantages of the latter (cephalalgia, increased tension of the eye, etc.). In the same way

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[1] Transactions of the American Ophthalmological Society, vol. iii. p. 400.

[2] Moyen simple de reconnaître l'amaurose unilatérale simulée. Journal des sciences médicales de Lille, 1882, p. 870.

[3] Lehrbuch der Augenheilkunde, S. 478.

[4] Contribution aux moyens propres à démasquer la simulation de l'amaurose et de l'amblyopie monolatérales. Nová Lekarz, 1893, 1.

[5] Diagnose medico-legale militare della amaurose e della amblyopia monoculare. Giornale medico del regio esercito et della regina marina, 1887, p. 897.

reading at the near point may be made difficult by the instillation of a few drops of a solution of atropine between the lids of the normal eye, that is, if the fellow eye is really affected with blindness. In all cases it is necessary to take note of the state of refraction.

*Cylindrical Glasses.*—The use of cylindrical glasses which have been extolled by Kugel, Lippincott, Ségal, etc., is far from giving, in the writer's opinion, very definite results. It is proper, naturally, to be assured that the eyes to be examined are not affected in any degree with astigmatism.

*Kugel's Test.*[1]—The first experiment intended to unmask the simulation of unilateral blindness consists in making the patient look at a point of light furnished by a candle which burns in a box closed on every side except on a level with the anterior wall, at the middle of which there is an opening of three millimetres in diameter which is fitted with a glass.

A patient having normal sight sees this point of light in the form of a luminous cross if cylindrical lenses with the axes perpendicular to each other are placed before the two eyes; while under the same conditions, a person affected with unilateral amaurosis will see but one luminous line, which is vertical or horizontal in accordance with the direction of the axis of the cylinder.

The following tests indicate to a certain degree the amount of visual acuity. A cylindrical lens which prevents the eye from seeing sufficiently to enable the subject to count a number of parallel lines traced on a piece of paper is placed before the normal eye and held at a certain distance. If the patient succeeds in counting them, it can be accomplished only with the eye that he has declared to be defective.

Again, the experiment can be made in the following manner: the two eyes being furnished with cylindrical lenses, the axes of which are perpendicular to one another, the patient is made to look at some crossed vertical and horizontal lines which can be seen clearly only when both eyes possess a normal acuteness of vision. If one of the eyes is amblyopic, it cannot be made to distinguish the lines that are perpendicular to the axis of the cylindrical lens with which it is furnished, this being alone possible with a normal eye. If, therefore, in this test, the patient counts the two kinds of lines equally well, the imposture is exposed.

*Lippincott's Test.*[2]—This test depends upon the trapezoidal distortion of the images that are produced, when binocular vision exists, by placing a two-diopter cylinder lens with its axis at ninety degrees before one eye. A two-diopter cylinder with its axis vertical is placed before the healthy eye, and the patient is made to look at a book or a visiting card. If he states with precision that the card or book has one side longer than the other, it is proof that he is seeing with both eyes.

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[1] Ueber Diagnose der Simulation von Amaurose und Amblyopie. Wiener medizinische Wochenschrift, 1889, Nr. 6, 7, 8 und 9.

[2] New Tests for Binocular Vision. Transactions of the American Ophthalmological Society, 1890, pp. 560–565.

Ségal[1] places a convex cylinder of one diopter strength before each eye. The axis of one is arranged at right angles to that of the other. If reading is as easy with both eyes as with the normal eye alone when not fitted with glasses, it may be concluded that the case is one of simulation. Strong cylindrical glasses, through which the patient is made to look at the flame of a candle, can also be used.

Before the good eye Jackson (*loco citato*) places two cylinders with their axes at right angles to one another, covering the suspected eye with a correcting lens. While the patient is reading one of the cylinders is rapidly rotated, and thus the healthy eye is excluded from vision.

*Prisms.*—The prism occupies an important position among the means of detection. It is inexpensive and its employment is very simple. It allows the physician opportunity to vary the experiments, and not only to detect simulation of blindness and unilateral amblyopia, but also to determine the presence of concentric contraction of the visual field (Schmidt-Rimpler).[2] It is well known that a prism has the property of deviating rays of light towards its base. Placed vertically before one of the eyes it annuls binocular vision in the vertical meridian. The perception of a double image will reveal, therefore, the power of seeing with each eye, and frequently also, if a letter of definite dimensions be used, it will give the visual acuity of the pretended blind eye. Such is the point of departure from the classical method of von Graefe, the first in order of priority (1885).[3] To carry out this experiment with success, it is expedient to question the patient in a special way and in such a manner as to give him the impression that his thoughts are understood.

Instead of being asked, for instance, if he sees two images, which would give him an opportunity of replying in the negative, he must be requested to state at once if the two images are one above the other; if they are placed obliquely; if the colored image is above or below; and if it is to the right or to the left. Thus confused, the malingerer will answer the question, so put to him, in a definite manner.

1. In 1867, Alfred Graefe[4] modified this method with advantage by giving the malingerer in the first place the impression that it is possible to see double with one eye alone, and by converting, unknown to him, a monocular into a binocular diplopia. For this purpose, after the pretended blind eye is closed, either the edge or the base of the prism is placed before

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[1] Contribution à la découverte de l'amaurose simulée. Vestnik oftalmologii, 1895.

[2] Schmidt-Rimpler, Zur Simulation concentrischer Gesichtsfeldeinengung mit besonderer Berüksichtigung der traumatischen Neurosen. Deutsche medicinische Wochenschrift, 1892, S. 561.

[3] A. von Graefe, Ueber ein einfaches Mittel, Simulation einseitiger Amaurose zu entdecken, nebst Bemerkungen über die Pupillarcontraction bei Erblindeten. Archiv für Ophthalmologie, 1855, S. 266

[4] Simulation einseitiger Amaurose. Klinische Monatsblätter für Augenheilkunde, 1867, S. 53–59.

the sound eye in such a way as to cover only a part of the pupil, and thus produce a monocular diplopia. The prism is then moved in such a way that it covers the whole pupil while at the same time the other eye is uncovered. If the double image remains, the malingerer thereby admits that he is seeing with the two eyes. Since 1881 the writer has particularly insisted on the relative difficulty of provoking monocular diplopia by the edge of a prism, while nothing is easier than to produce it very distinctly, and without feeling one's way, by using the base of the prism.[1]

2. *Galezowski's Birefractive Prism.*—With the object of producing monocular diplopia with greater certainty, Galezowski utilizes the property of double refraction which Iceland spar possesses, and produces alternately double monocular and binocular vision, by placing successively in front of the eye a double refractive lens of Arago, and the ordinary prism which cannot be distinguished from this lens by external appearance.

The use of a double refractive lens presents the advantage, as in the writer's method, of assuring the person examined that he certainly sees double, but it presents several inconveniences, among others a perceptible difference in the images.

Nothing is simpler, as Frölich[2] points out, than to produce alternately monocular triple vision, and binocular triple vision by means of a double refractive prism of fourteen degrees, handled in a manner that is similar to the way employed for the simple prism in the two tests that constitute the Alfred Graefe test.

3. *Monoyer's Double Prism.*—Monoyer uses two prisms of ten degrees each. These are united by their bases, which, by means of a special mechanism, can be separated from one another to the extent of one millimetre's width. By this contrivance the expert can produce, at will, simple deviation or monocular double or triple vision, according as he makes one or the other of the prisms, the line of junction of their bases, or a portion of the bases of both prisms with the interval which separates them, lie in front of the pupil. Monoyer's double prism is a very ingenious apparatus, but it is more complicated as a mechanism and as a method than the simple prism. In monocular diplopia it produces images differing in intensity, in color, and especially in the degree of separation from those which binocular diplopia gives. In fact, the separation of the images in monocular diplopia produced by the junction of the two bases is double that of the images that are obtained by the single deviation (action of one prism alone) in binocular diplopia. Moreover, it allows the malingerer to take note of the movements which the examiner causes the prism to make

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[1] Simple note sur l'emploi du prisme pour provoquer la diplopie monoculaire. Application à la recherche de la simulation. Bulletin médical du nord, pp 539–548, 1881. Sur l'emploi du prisme comme moyen de dévoiler la simulation de la cécité unilatérale. Archives d'ophthalmologie, 1882, pp. 10–22.

[2] Prismen und erheuchelte einseitige Blindheit. Klinische Monatsblätter für Augenheilkunde, 1895, S. 263–272.

during the experiment, although the mechanism of the instrument may be concealed, as far as possible, in a metal box.[1]

4. *Frölich's Method.*—In the opinion of Monoyer, it might be advantageous to modify the construction of Frölich's apparatus by employing different colored glasses in such a way as to baffle the malingerer, and to enable the examiner to control all statements. Frölich appears to have profited by this idea in adding to the double prism a red glass, which by a special mechanism can be adjusted sometimes before the two prisms placed base to base, sometimes before the space which separates them, and sometimes before one or the other of the prisms. The three images, the upper and the lower, or the single middle image, can thus be colored red at the will of the examiner. The apparatus is still more complicated than that of Monoyer; in the fact that the second red glass, which is placed before the eye that is said to be defective, must destroy the incessant surveillance that is indispensable to exercise in order to prevent a malingerer from closing the eye and discovering the number and color of the images which it is to his interest to declare that he sees or does not see.

In the use of prisms and of the methods that are derived from their employment several causes of error are inherent. Among the most important may be cited: *A.* The perceptible difference in clearness and color between the real and the false image, the borders of the latter being iridescent. The false image of the candle-flame, given by a birefracting prism, is, in particular, much less brilliant, since the incident light is divided into two bundles that are refracted with equal intensity by each and with half the intensity of the incident rays. *B.* The special shape of the glasses used, allowing the malingerer to see that sometimes the base or the edge, sometimes the prism itself, or that sometimes the double prism, has been placed before the eyes. The possibility of noticing the displacing movements which the examiner makes during the experiment, in order to make binocular double vision follow monocular double vision, is another source of error in their use.

5. *Baudry's Method.*—The method which the writer proposes does away with, almost completely, these various inconveniences, and obtains a series of double images that are so similar that the malingerer cannot distinguish the false from the real one, or discover whether the double vision is the effect of a monocular or of a binocular diplopia. Moreover, the arrangement of the instrument which is used for the experiment is such that the malingerer cannot previously ascertain whether he has the base only of the prism or the whole prism placed before the eye that he has declared to be sound, even if he is acquainted with the mechanism of the apparatus. For this test, a dark red glass of even color is placed before the flame of a candle

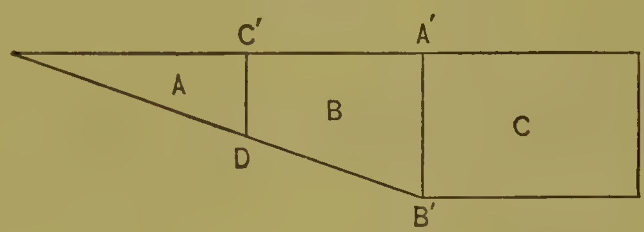
[1] Monoyer, Note sur trois nouveaux moyens découvrir la simulation de l'amaurose et de l'amblyopie unilatérales. Gazette hebdomadaire de médecine et de chirurgie, 1876, p. 388.

which is situated at a distance of two or three metres. Here the color of the false image, being produced by the decomposition of the white light in its passage through the prism (if, instead of white light, we use red light, —such as passes through a red glass colored by oxide of copper), cannot undergo any further decomposition, and, as a result, the *real and the false images are identical* in appearance. The interposition of this dark red glass renders the difference which still exists between the images, in binocular and in monocular diplopia, scarcely perceptible.

The instrument which is used has the following arrangement: A triangular prism, on section a right-angled triangle, divided into two parts by a line of horizontal section, C′D, is united by its base, A′B′, to a transparent medium, C, with parallel surfaces and of the same thickness. The whole glass represents a portion of a bevelled mirror without the mercury, divided into three distinct parts, A, B, C, which lie with their unpolished cut surfaces in apposition. (Fig. 1.)

This glass is concealed in a circular metal box (oxidized brass) which is perforated on each surface by a central opening, one of which has a diameter of six and the other of three millimetres. A simple mechanism which allows sometimes one and at times the other of the two lines of separation (A′B′ or C′D), and at the same time a small part (three millimetres) of the adjoining portions of the glass (or, in an optical sense, sometimes the base of the prism and sometimes the prism itself) to be brought before the pupil of the sound eye, is thus obtained. As the lines of division and the adjoining portions of the glass that are brought before the pupil are absolutely identical in appearance, sometimes monocular and sometimes binocular diplopia, unknown to the malingerer, even if he knows in advance the construction of the instrument, can be evoked with the greatest ease.

FIG. 1.




It is almost superfluous to point out the different methods of applying this test. The person examined being permitted to believe that the examiners are convinced of the reality of his disease, the eye that is supposed to be blind is, without exercising any pressure, covered by the examiner's hand, and the patient is made to fix his gaze on the flame of the candle that is placed two or three metres away behind a sheet of dark red glass. The instrument is then arranged in front of the normal eye, the line of separation between the base of the prism and the medium with parallel surfaces, A′B′, cutting horizontally the area of central aperture, which is placed in line with the pupil. Under penalty of showing evidence of bad faith, the patient will assert that there are two images of the candle-flame. On withdrawing the instrument, the line of separation of the two portions of

the prism, C′D, is brought instantaneously, and without the knowledge of the patient, in place of the preceding, A′B′, and the instrument is replaced before the healthy eye, though the eye that has been declared to be blind is purposely left uncovered. The patient declares that there are still two images. He is therefore completely deceived, since a monocular diplopia has been replaced by a binocular diplopia.

It may be that the patient has taken the line of denying obstinately the existence of double vision. In this case the two parts of the test can be repeated several times, in order to catch the malingerer in a mistake. The writer maintains that these modifications of the prism-test are of a nature to baffle the most intelligent and the best-informed patient, since, on the one hand, owing to the interposition of a dark red glass, the two images resemble one another, in monocular as in binocular diplopia, and, on the other hand, with the instrument, the interested person cannot distinguish whether he has the base of the prism, or the prism itself, in front of the eye that has been declared to be normal.[1]

Alfred Graefe has pointed out the means of ascertaining by the aid of a prism the feigning or exaggeration of a unilateral amblyopia. It is sufficient to place in front of the sound eye a twelve-degree prism, with its base upward, and to make the patient look at letters that have been cut from a printed scale and pasted on a piece of white paper. The words or letters are thus seen double and placed one above the other; the patient, by reading the upper line as well as the lower one, determines, unknown to and in spite of himself, the degree of visual acuteness of the affected eye. The difficulty in succeeding with this test arises from the fact that the interested person can take into account the difference in the clearness of the images (one set being weakened by the effect of the prism), and can arrange his answers to suit himself.

Finally, one of the principal advantages of the prism is that, in the absence of the stereoscope, the same experiments can be reproduced and varied in several ways with this inexpensive glass.[2] For such a purpose all that is necessary is to place the prisms in a trial frame, with the base placed sometimes up, sometimes down, sometimes in, and sometimes out. These combinations of the position of the prisms will infallibly baffle a patient, who will believe that he sees certain letters with one eye, while in reality it is with the other; his contradictory answers causing the discovery of the imposture.

With this train of ideas, Ohlemann recommends a test which has enabled him to rapidly detect malingering. It consists in placing, at first, before one eye a prism of about ten degrees, base down, and making the

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[1] Baudry, Démonstration d'un procédé facile et certain de provoquer la diplopie monoculaire à l'aide du prisme simple. Son application à la recherche de la simulation de la cécité unilatérale. Douzième Congrès internationale de Médecine de Moscou, August, 1897.

[2] Schmidt-Rimpler, Zur Erkennung der Simulation von Blindheit. Klinische Monatsblätter für Augenheilkunde, xiv. S. 178.

patient look at a light at a distance of some metres. The patient will then perceive two images. Arranging then in the same way another prism of the same strength before the second eye, the patient is asked if he still sees two images. A reply in the affirmative indicates malingering. The experiment can be continued by varying the position of the prisms in different ways, and thus deceiving the examined person.[1]

The writer pointed out above a minor desideratum in the employment of the Javal-Cuignet method,—that is to say, the necessity of absolute immobility of the patient's head. In tests with prisms it is an *absolute immobility of the glasses* which is indispensable, for the least movement of one of them will produce a displacement of the corresponding image, making it comparatively easy for an experienced malingerer to conduct himself accordingly.

6. *Baudon's Method.*[2]—Baudon uses two prisms of about fifteen degrees, the one red, and the other blue. These are placed in front of the eyes, bases out, and the patient is made to look at a candle-flame that is situated two metres away in a dark room. If the malingerer declares there is only one candle, it will be the one which he thinks he sees with the sound eye, while in reality it is the one that is seen with the eye which he pretends is blind.

On the same principle Billot and Baudon have constructed an apparatus, made like a stereoscope, which has the appearance of an opera-glass, and which, in the case of amblyopia, enables the visual acuity of the eye that has been declared defective to be determined. The apparatus is made of two tubes, each of which is twenty-five to thirty centimetres long, the eye-pieces being provided with prisms of twenty degrees, bases out. As test-objects, reproductions of the letters of Snellen's scale are used.

7. *Schenkel's Method.*—This consists in placing before the two eyes of the patient two sixteen-degree prisms, with the edge of one up and of the other down. He is then requested to count the parallel lines that form a part of Snellen's types, and then to touch with his finger the upper and the lower lines. This he will be unable to do if he is not really affected with unilateral amaurosis.

8. A prism with the base vertical can be placed before one of the eyes, and the patient asked to go up and down a staircase with which he is not familiar. This act will be very difficult unless he closes one of his eyes.

9. While constantly rotating a prism of twenty degrees before the *right eye* the patient is made to read aloud. If binocular vision exists, reading letters or deciphering very fine signs will be difficult, or at least the patient

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[1] Ueber Aggravation bei Augenverletzungen (Amblyopia vera et spuria), Zeitschrift für Medicinalbeamte, 1893, 143.

[2] Note sur quelques moyens pratiques destinés à reconnaître l'amaurose et l'amblyopie simulées. Description de deux procédés nouveaux. Recueil d'ophtalmologie, 1877, p. 278.

will hesitate, while the really one-eyed will be in no way incommoded (Berthold's method).[1]

#### 4. TESTS WITH THE STEREOSCOPE.

To Z. Laurence, according to some, and to Hogg, in accordance with others, belongs the idea of using the stereoscope for the detection of feigned monocular blindness.

Whether the ordinary cased stereoscope, the American, the horizontal, or the open stereoscope, or any improvised apparatus is used, the principle of the instrument is always the same. There are two prisms, placed bases out and apex to apex ; and it is chiefly the prismatic action of the stereoscope which is turned to profit for the production of the different images by their superposition, their fusion, their displacement, or their intercrossing.

In a general way, the use of the stereoscope, already difficult when the two eyes are not of the same refraction, demands as an essential condition of success the integrity of binocular vision. Another inconvenience that is inherent in the different methods is the difficulty in preventing the malingerer from momentarily closing one of the eyes (which makes the test ineffective), and from his being able to realize the situation of the objects, owing to the prismatic coloration or to the differences in the clearness of the images. A person who has normal vision in the two eyes, and who looks through the prisms of a stereoscope, fuses and involuntarily superimposes the two lateral images into a single combined image. It is in this way, for instance, that Fig. 2 will assume the appearance of Fig. 3 in the stereoscope when suitably placed on the same card. Again, a sentry box on one side and a soldier on the other can be arranged so that a person looking at them through a stereoscope will fuse them binocularly and will see the soldier in his sentry box.

Fig. 2.

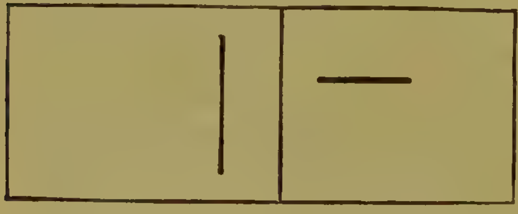


Fig. 3.

There are some persons who do not immediately succeed in fusing the images, and, in consequence, they appreciate the situation. Others who are anisometropic, or are amblyopic in one eye, are said to always see double images. To guard against these inconveniences experiments have been devised in which the displacement or intercrossing of the images produced by the prisms is utilized.

A very simple arrangement consists in placing two wafers, one red (1) on the right and the other black (2) on the left (Fig. 4), one centimetre from the vertical line which divides the test-card into two equal parts and which

[1] Ein neues Verfahren, die Simulation monoculärer Blindheit zu ermitteln. Klinische Monatsblätter für Augenheilkunde, 1869, S. 300.

corresponds to the partition of the instrument. The malingerer, being previously unconscious of the intercrossing, will think that he sees on the right side what ought to be seen on the left, and will betray himself at the outset (Vieusse).[1]

A better method is the following. On a stereoscopic chart, on each side of the median line, four wafers are pasted. (Fig. 4.) The two upper ones, red on the right (1), black on the left (2), are situated at one centimetre's distance apart. The two lower ones, yellow on the left (3), blue on the right (4), are five centimetres distant. There will be, as a result of this disposition, a crossing of the upper and approximation of the lower wafers.

Fig. 4.

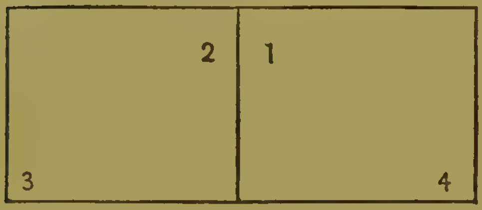
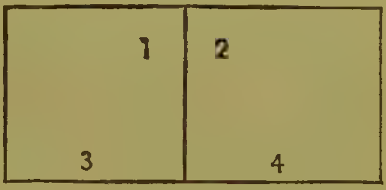



Fig. 5.




(See Fig. 5.) The malingerer, if he does not close one of his eyes during the experiment, will be at a loss to designate the wafers which are on his right and those which are on his left. Nothing is easier than to arrange a chart to vary the number and the position of the wafers, and to replace them by pictures or signs for the illiterate, by figures, or by words that are susceptible of transposition.

Other methods have the advantage of unmasking the fraud and indicating at the same time the degree of visual acuity.

*Monoyer's Stereoscopic Tests.*—Let it be supposed that there have been constructed on each half of a stereoscopic test-card similar letters, arranged identically in the same order, and separated by similar intervals in the two tests in such a manner that they are faithful fac-similes of one another without the slightest difference of stereoscopic parallax. Further, let it be imagined that a certain number of letters or entire words, or even fragments of letters, selected hap-hazardly, have been omitted from each half of the test-card, care having been taken that the omissions on one of the test-cards do not coincide with those on the other: the site of the suppressed portions being left blank.

In the same way ten charts with letters progressively increasing in size, and so calculated as to represent the ten degrees of the decimal typographic scale, can be prepared, and made to serve for measuring the acuteness of sight in tenths of a unit. Several sizes can be brought together on the same chart.

These charts being placed in turn in the stereoscope, the malingerer is asked to read the words or to spell the letters which he sees, beginning with

[1] Amaurose simulée et le moyen de la découvrir à l'aide d'un stéréoscope. Recueil d'ophtalmologie, 1875, p. 248.

the largest size, which corresponds to $V=1$. The number of the letters which have been read combinedly on the two halves of the chart, will at once reveal the visual acuity of the amblyopic eye. As an extra precaution, it is convenient to have, in addition, a chart, the two halves of which are identical, and to primarily make the test with it, in order that the person who is subjected to this method of examination may not suspect the methods that are applied to unmask a fraud.[1]

As having more particularly tended to perfect the stereoscopic test, attention will be called to the arrangements that have been contrived by Rabl-Rückardt,[2] Burchardt,[3] Schröder,[4] etc.

The American stereoscope, introduced into practice in 1873 by Rabl-Rückardt, has the advantage of facilitating the constant surveillance of the patient's eyes. At the least attempt at closure of the lids, the physician —thanks to the sliding chart of Burchardt, or to the sliding apparatus of Schröder—can put the most artful malingerer off his guard by consecutively showing him a negative card or a test-chart, while the two eyes are open.

Very ingeniously combined tables and scales, especially prepared for stereoscopic tests, have been published; such are Kroll's scales, which are composed of words and figures that are susceptible of transposition, with symbols for the illiterate. C. Hoor's tables with colored disks and Ségal's stereoscopic plates are also useful.

Hoor makes the objection to the different forms of stereoscopic scales or pictures, which appears to the writer to be well founded, that they are composed of letters or signs having on the two sides the same size, the same thickness, and the same intensity of black color. The result is that if the malingerer has one eye much weaker than the other, the letters will appear to this eye less clear and dimmer, and will thus allow him to realize the actual condition of things, and to distinguish what is to his interest to declare that he sees or does not see.

In order to obviate this inconvenience, Hoor proposes to select for test-objects colored disks of variable size, and of alternately darker and lighter shades on one side than the other, so as to prevent the individual from obtaining information from such a source. The degree of visual acuity can also be very approximately calculated from the dimensions of these disks.[5]

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[1] Monoyer, Note sur trois nouveaux moyens de découvrir la simulation de l'amaurose et de l'amblyopie unilatérales. Gazette hebdomadaire de médecine et de chirurgie, 1876, p. 388.

[2] Ueber Simulation einseitiger Amaurose. Deutsche militärärztliche Zeitschrift, 1873, Heft 1. Ueber Vorteüschung von Blindheit. Vierteljahrschrift für gerichtliche Medicin, etc. Neue Folge, Band xxiv. S. 74–96. Zur Entlarvung der Simulation einseitiger Blindheit durch das Stereoscop. Berliner klinische Wochenschrift, 1884, S. 83.

[3] Praktische Diagnostik der Simulation von Gefühlslähmung, von Schwerhörigkeit und von Schwachsichtigkeit, Berlin, 1891.

[4] Zur Frage der Aufdeckung der Simulation einseitiger Blindheit. Berliner klinische Wochenschrift, Nr. 44, 1883.

[5] Neue stereoscopische Tafeln zur Constatirung simulirtir monoculärer Amblyopien und Amaurosen. Der Militaerarzt, 1889, Nr. 11, 12.

Ségal (*loco citato*) has suggested an ingenious means of detecting feigned amaurosis, which is based on the principle of the mixture of colors and prismatic action and the functions of the stereoscope. If stereoscopic plates are constructed in such a manner that the letters or symbols (or one of them), arranged symmetrically on the two sides, are of a definite color on the right side and of a complementary tint on the left, these letters or symbols, when fused, will appear grayish white in the stereoscope. New color effects, which the malingerer may even be familiar with beforehand, if non-complementary colors are used, can be obtained in the same way.

Straub has converted the apparatus of Gratama into an instrument which combines at the same time the action of the stereoscope and that of the pseudoscope.[1]

The original instrument of Gratama is made of two parallel tubes, provided at both ends with sliding plates, that cut off a part of the width of the tubes, and in which there are rectangular apertures. Beyond the intersection of the two visual lines a printed scale is placed in front of one of the tubes and a white card in front of the other. The transposition of the images being obtained by the intercrossing of the visual lines, the malingerer reads with the eye that is declared to be amblyopic what he thinks he is reading with the sound eye. To this apparatus have been added two three-degree prisms, placed base inward inside the tubes near their ocular ends in such a way that the superimposed letters form words. This test presupposes, it is unnecessary to say, that the person examined enjoys binocular vision.

Finally, the stereoscope-optometer of A. Baldanza[2] is a box similar to that of the ordinary stereoscope, with a mirror hinged to the upper wall in order to illuminate the test-types that are placed at the end of the apparatus. It is about thirty-three centimetres long, five by nineteen centimetres broad, and nine centimetres high; the two latter dimensions gradually lessening towards the anterior part of the box, in which situation they are not greater than twelve and five centimetres each. The eye-pieces are provided with four twelve-degree prisms. The two front ones are stationary, with their bases placed outward, while the two others, placed exactly behind, are movable in such a way as to double or neutralize the refracting power of the front ones. A vertical screen divides the box into two halves, so that the types that are placed on the right cannot be seen by the left eye, and *vice versâ*.

The arrangement of the prisms allows a series of combinations that are capable of baffling the most cunning malingerer, even if he is familiar

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[1] De toestel van Dr. Gratama voor de herkenning van voorgewende gezichtszwakte op een oog. Nederlandsch militair geneeskundig, Archief van de Landmacht, etc., 1888, xii. S. 409–414.

[2] Un nuovo mezzo di misura dell' acuita visiva per i sospetti simulatori dell' amaurosi o della amblyopia monoculari. Giornale medico del regio esercito e della regina marina, 1897, p. 376.

with the mechanism of the apparatus. Moreover, the lack of clearness of the deviated images can be gotten rid of, since the neutralization of the prisms allows them to be seen as through a plane glass.

### 5. TESTS BY COLORED GLASSES AND LETTERS.

Kugel,[1] in order to take the malingerer unawares, has advised putting before his eyes blue glasses, one of which is opaque. The opaque one is placed before the sound eye, thus preventing the patient from seeing anything with the good eye. If the patient does not perceive the deceit and reads letters which are shown him, the deception becomes evident, and visual acuity is approximately estimated. A certain number of excellent methods (when the patient is not well informed) to detect the feigning of amaurosis and amblyopia are based on the application of the physical laws which govern the absorption of light in its passage through refractive media (methods of Snellen, Rava, Dujardin, Stoeber, Bravais, Fontorbe,[2] Michaud, Minor, etc.).

It is known that a plate of glass of definite color, and which is monochromatic, allows similarly colored rays to pass through it, and stops rays of the complementary color; consequently it does not allow letters or symbols that are printed in the complementary color to be read. As a result, if vision is attempted through a red glass, for instance, for red and green letters or symbols that are printed on a black surface, the red will be seen clearly, while the others, having become black, will be confounded with the surrounding black surface and will disappear. If the groundwork is white, the red letters, by reason of the interposition of the *red* glass, will become invisible, and the green ones will appear *black.*

While pretence is being made not to doubt the patient's statements, he is to be shown at five metres' distance a chromatic table having on a *dead black* surface alternately placed *red* and *green* letters, which cannot be distinguished from the surface by glistening. Under the pretext of being sure that the sound eye works normally it is provided with a *red* glass. The reading of the *green* letters will be an evident proof of untruthfulness, and at the same time the visual acuity is measured. Instead of the red glass, a green glass through which the red letters will be invisible can be used. (Method of H. Snellen.[3])

Letters produced by printing or by lithography are not very well adapted to the experiment with colored glasses. The glistening of the letters or of the colored symbols has not infrequently enabled the malingerer to distinguish them from the groundwork of the plate. For this reason it has

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[1] Ein Methode, um in leichter Weise Simulation einseitiger Amaurose und Amblyopie zu constatiren. Archiv für Ophthalmologie, 1870, S. 343.

[2] Bastier, Examen de la vision pour le service de la marine. Thèse de Montpellier, 1888, p. 57.

[3] Entdeckung der Simulation einseitiger Blindheit durch Prüfung mittelst farbiger Sehproben. Klinische Monatsblätter für Augenheilkunde, 1877, xv. S. 303.

been suggested, first, to substitute for the Snellen and Stilling scales, the Pflüger plates, the letters of which, being printed in different shades of red, give the impression of green by the interposition of tissue-papers; second, to replace the letters in chromolithography by transparent characters made on colored glass; and third, to trace with colored chalks figures, letters, or symbols on black paper, or on black plates, avoiding heavy pressure (Michel).

Whatever be the method that is employed, it is essential, before beginning any experiments, to see that the colored letters or symbols are not apparent, by glistening, on the surface of the plate; that the tint of the letters is appropriate to that of the colored glass; that the plates or scales are placed well in front of the light and are perpendicular to the direction of the visual lines; and, finally, that the tests are repeated upon one's self with both eyes open, then with one eye shut, and while the other is provided with a colored glass.

Stoeber[1] has constructed, on the same principle, a portable scale that is composed of six squares of red and green glass of the same size, on each of which there is a black letter, selected from the decimal scale of Monoyer. These squares of glass are arranged in threes in two horizontal rows, and are supported on two sheets of card-board that are joined by a hinge which allows them to be applied one against the other. In this way an instrument of small volume and of small net cost is obtained. If a pair of spectacles, of which one of the lenses (the left) is red and the other is green, is placed before the patient's eyes, it is evident that it will be impossible for him to read the red letters, if the right eye is really blind.

Rava[2] has constructed a rectangular box with its posterior wall made *red*, and with the upper wall composed of ground glass. The other walls are blackened. The front one is perforated with two openings that are closed by two ordinary glasses. A slide allows a plate of green glass to be passed in front of one or the other eye-piece, rendering it possible to ascertain if an eye is really amaurotic, according as the end of the box appears red or coffee-black in tint.

Having considered that red glasses render letters of the same color traced on *white paper* invisible, Bravais[3] and Dujardin have pointed out the simple and very practical method of substituting the tables of Snellen and Stilling. The patient, upon whom a spectacle-frame, provided with a red glass on the left side, has been adjusted, is placed before the ordinary test-type, and can be made to take note by closing the right eye (which is supposed to be blind), whether the glass that is placed before the sound

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[1] Echelle pour déterminer la simulation de l'amaurose unilatérale et l'acuité visuelle. Archives d'ophtalmologie, 1883, p. 267.

[2] Novello metodo diagnostico per iscoprire la simulazione de l'amaurosi monoculare. Annali di ottalmologia, 1881, x. 289–291.

[3] Simulation de l'amaurose unilatérale. Nouvelle forme donnée à l'épreuve par les verres coloriés de Snellen. Bulletin et mémoires de la Société français d'ophtalmologie, 1884, p. 166.

eye modifies the vision in any respect. If, at the moment of the examination, one or more words in two colors (red and black letters) are traced on white paper, the first (red) will be invisible if the right eye is really amaurotic; the reading of entire words or of all the words of a phrase being evident proof of malingering (Dujardin).

Bravais has modified the Snellen method in a way which enables him to dispense with the chromatic scales and to vary the tests in different ways. He places before the sound eye a *red* glass, and before the one that is declared defective a *blue* glass. He then has the patient read some letters or words drawn on a *white* sheet of paper with *red* and *blue* chalk. The letters are arranged alternately blue and red, in such a way as to form words of totally different meaning, according as the person examined sees with one or with both eyes. For example: "Chart" will become "Cat," and "I don't see very clearly," "I see clearly."

The usefulness of the employment of blue glass and blue chalk depends on the following reason: With blue glass the red letters appear darker than they would to the naked eye, and the blue ones grow pale, so that the former, which are effaced for the sound eye, and which without a glass would be a great deal paler than the blue, become, on the contrary, for the suspected eye, with the co-operation of the blue glass, more plainly visible; so that, if this eye is not amaurotic, the patient will call these letters red as well as those which he sees with his good eye.

The combination can be further varied by writing the letters with a black pencil on charts that are half red and half blue. Each eye can then see only certain letters, since the writing on the blue ground is not visible through the red glass, while that on the red ground is not visible through the blue one.

*Michaud's method*[1] has, perhaps, more chances of troubling and taking the malingerer by surprise. It consists in constructing scales for distant-vision and for near-vision, of letters composed of strokes or hangers (limbs) of different colors (*red and black*), and drawn with a pencil on so-called architect paper which has been ruled both ways by millimetre divisions in red-brown, in such a way as to give the letters dimensions that are similar to those of ordinary test-types. If, for instance, to the letter "I," painted black on *white paper*, red horizontal lines are added, the letter "F" or "E" or "L" or "P" is obtained, etc., according to the relation of the red to the black lines. With these bicolored letters it is easy to make words, the appearance and meaning of which will be different from that of the same letters that are formed only of black strokes. The sound eye being provided with a red glass, under the pretext of making sure that the colors can be all distinguished well, the patient is induced to read the letters of the chart quickly. If the other eye is really amaurotic or amblyopic, the

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[1] Procédé pour reconnaître la simulation de l'amaurose et de l'amblyopie monoculaires devant les conseils de révision. Archives de médecine et de pharmacie militaires, 1888, No. 4, p. 264.

normal one will only be able to distinguish the black letters. If the contrary be the case, the malingerer will read the bicolored letters, and their dimensions will give the degree of visual acuity of the eye which is pretended to be blind.

With the object of preventing the patient who is acquainted with the method from distinguishing easily all of the red letters and from omitting them from the answers, Mullier has advised retouching some of these red letters very lightly with a black pencil.[1]

Finally, Minor[2] has very ingeniously combined Snellen's letters as seen through a glass of complementary color with green and red letters placed on opaque gray surfaces. The shades of these letters are chosen in such a way that, seen across a glass of similar color, they cannot be distinguished from the gray opaque surface. Williams[3] has proposed an analogous proceeding,—red and green letters on a gray ground, which are to be seen through red and green glasses, etc. It is necessary to insist on a previous examination of the color-sense to be assured that the patient is not affected with achromatopsia or dyschromatopsia, either congenital or accidental, in order to avoid error. For this examination Holmgren's skeins of colored wools, Stilling's chromatic charts, or by preference the charts of Pflüger, based upon the phenomena of contrast, may be used. If the answers of the patient cause a suspicion of malingering in this respect, recourse may be had to a mixture of different colors, or the patient may be made to look through a colored glass. For instance, if the patient pretends to confound red and green, he may be made look at the charts through a red glass; and as the green rays do not pass through the glass, the red ought to appear lighter than the green.

## II.—FEIGNED UNILATERAL AMBLYOPIA.

It is much more convenient for the malingerer to declare that he has sufficient sight to guide himself and to distinguish large bodies, but that he does not see clearly enough to work. Every practitioner of moderate experience admits the frequency of cases of the feigning of pronounced amblyopia following insignificant ocular injuries. The writer has dwelt at length on this point in another work.[4] Frequently the injured person, who has lost one eye, refuses to resume work under the pretext that the sight of the fellow-eye is considerably weakened since the injury. Sympathetic amblyopia may certainly exist, but it would be an exaggeration to infer the presence of this complication in every case in which the other eye had been previously injured.

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[1] Van der Straeten, Des moyens de reconnaître la simulation de l'amaurose et de l'amblyopie. Archives médicales belges, 1892, p. 241.

[2] New Colored Letter-Tests for Simulated Monocular Blindness. Archives of Ophthalmology, xxii. 1893, 493–495.

[3] British Medical Association, Montreal, 1897.

[4] Étude médico-légale sur les traumatismes de l'œil et de ses annexes, Lille, 1896.

In Germany in particular, since the putting in force of the law relating to accidents and *caisses d'invalidité*, malingering has made a considerable advance. Seeligmüller[1] and Fr. Schultze[2] give an average ratio of twenty-five to thirty per cent.

Workingmen, as Nieden[3] so justly writes, do not consider the indemnity which is due to them a just compensation for the injury caused; they seek to create for themselves resources through the accident of which they have been the victims. With this object they have a tendency to prolong the effect of their injury in order that the sum of the indemnity may be increased. They exaggerate the slightest accident, and even feign visual troubles (asthenopia, etc.) which do not exist. This has become so bad since the promulgation of the insurance law of 1884, it has been ascertained that in the district of Saarbrück there has been an increase in the number of cases of illness due to accident in the proportion of twenty-five per cent. The duration of treatment has increased in the proportion of thirty per cent., while the total number of cases of spontaneous disease remains absolutely the same.

Rumpf had already pointed out the fact that formerly patients resumed work more rapidly after serious wounds than they do to-day after slight injuries. If to this is added that the address and astuteness of malingerers has increased with their number, and that the cases are sometimes difficult to determine, it will be seen how much more difficult it is for the physician to decide if the amblyopia is real or feigned. It also becomes hard to know to what extent it is exaggerated; whether it is exclusively of traumatic origin, and whether it is the result of an old malady. Moreover, it becomes difficult to decide if the causal injury is, indeed, that to which the injured man attributes the defect. It is also hard to know if the amblyopia did not exist, unknown, previous to the traumatism. How many patients who come to consult the physician on account of a wound in an eye take notice at that time only of a visual defect which they did not previously suspect in the opposite eye, and which they are inclined to attribute to the traumatic lesion, which has had nothing to do with it. It is by dint of ingenuity and patience that the physician is able to catch the patient in contradictory answers; and yet the medical adviser's judgment cannot be formulated in all cases with absolute certainty.

Let a very strong convex lens be placed first before the eyes of the patient so as to enlarge the object as with a magnifying glass. If he declares that he does not see printed letters situated at a suitable distance more clearly,—that is to say, within the limits of the focal length of the

[1] Die Errichtung von Unfallskrankenhäusern. Leipzig, 1890.

[2] Weiteres über Nervenerkrankungen nach Trauma. Deutsche Zeitschrift für Nervenheilkunde, Band i. S. 545. Zur Lehre von den Nervenkrankheiten nach Unfällen. Deutsche medicinische Wochenschrift, 1892, Nr. 1 u. 2.

[3] Ueber die Simulation von Augenleiden und die Mittel ihrer Entdeckung. Wiesbaden, 1893.

lens, and objective examination of the refraction having shown that the eyes are emmetropic, for instance,—it becomes justifiable to suspect falsification. If the malingerer declares, on the contrary, that there is an improvement with a six-diopter convex lens, it may be suggested to him that the strength of the lens is to be doubled, and he thus becomes enabled to put the letters which he is reading farther away. The six-diopter lens is then neutralized with a concave lens of the same strength. If the patient continues to read the text, he indicates by his act the approximate degree of his vision. It will be the same if, on attempting to determine the visual acuity for distant objects at variable distances with letters of different size, and from one day to another, contradictory instead of identical replies are gotten. The establishment of a considerable disproportion in the results of testing near and distant vision is also suggestive. Far from suspecting the sincerity of the patient in these cases, it is necessary to take every means of gaining his confidence and then proceed to repeated and varied experiments.

Visual acuity being dependent on several factors, among which may be mentioned intensity of the illumination,[1] the adaptation of the eye to it, the size of the pupil, the refractive condition, the age of the patient,[2] the state of the accommodation, the general condition, etc., due allowance should be made for the influence of these different elements during the examination. It is for this reason that, when it is a question of proceeding to the examination of the visual acuity, it is indispensable to take into consideration the variations in the intensity of the illumination with diffused light. This is so, as light is most variable in our climate (H. Cohn), thus rendering it preferable to use artificial illumination, which can always be made uniform (Derby). If this cannot be done, the observer should measure his own visual acuity (Schweigger) in order to acquaint himself with the possible diminution that may be due to insufficiency of light, and to take it into consideration in the estimation of that of the patient. It is necessary, finally, to wait until the eye is adapted to the surrounding illumination. In this way can be explained, at least in part, by differences in the illumination, how such dissimilar results are obtained ; for example, visual acuity measured at different hours of the day (H. Cohn), or even at the same hour in the open air (Gaedicke), or in a room, even though it may be well lighted. The attempt has been made to remedy, in part, the insufficiency of the illumination by utilizing the transparent charts of Javal and of Cohn. These are placed against a window-pane and the patient sees their image reflected in a mirror that is arranged in front of the chart, thus allowing a working distance which is double that of the object, as in the method of Barthélémy. It should be remembered that differences in illumination

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[1] Von Kries, Ueber die Abhaen centraler und peripherer Sehschärfe von der Lichtstärke, Centralblatt für Physiologie, viii. S 694. Snellen, Notes on Vision and Retinal Perception, Transactions of the Ophthalmological Society, xvi., 1896.

[2] Katz, Contribution à l'étude de l'influence de l'age et l'acuité visuelle. Vestnik oftalmologii, November-December, 1896.

have less influence on eyes which possess normal vision than on those whose visual acuteness is diminished. With feeble illumination central vision in the hysterical patient diminishes to a greater degree than it does when the subject is in a normal state.

The use of various test-types, or of optometers, is far from being a matter of indifference. As the problem of a test-scale for universal adoption by ophthalmologists is not yet settled, and as the bases which serve for the construction of test-types present appreciable differences,[1] it is necessary to expect variable results in the determination of visual acuteness according as one or the other is chosen. It is also true that the amblyopic eye recognizes white letters more easily on a black ground than black ones on a white ground; that certain characters of the oldest and most universally used (Snellen's) scale, with certain symbols or designs for the use of the illiterate and children, are read more easily than others (Cattell). In fact, in spite of the precautions taken in the construction, the most recent charts still leave something to be desired in this respect. It is necessary to make an exception in the case of the decimal bracket ([ ]) scale, which has been perfected by Steiger, for the examination of distant and near vision, and the series of Pflüger, the reading of which can, besides, be made by reflection in a mirror, which is a matter that is worthy of consideration when only a small room can be used, and when it is a question of exposing a malingerer. The series of groups of points of different sizes, of which the international scales of Burchardt are composed (1893), are more appropriate for the determination of the punctum proximum and of visual acuity at short distances.

As for the charts of Guillery (black points on a white surface), of Wolfberg (colored points on a black ground), and the portable chart of Javal, they also permit the exact measurement of the visual acuity as with the decimal scales or Snellen's, and they have, besides, the advantage of serving as a means for the detection of malingering.

Practically, it is unnecessary to investigate the mathematical measurement of the visual acuity in any other way. Moreover, diminution of vision can be appreciated with sufficient exactness by adopting the same rules for examination with the same scale of characters.

With certain subjects, the trial with an optometer gives a degree of acuity which is superior to that obtained by Donders' method, but it has the advantage that, if the instrument be manipulated rapidly and quick replies are insisted on, the malingerer will more easily contradict himself. Most optometers, however, with the exception of those that work at a long distance, have the effect of exciting efforts of accommodation.

The dioptric state of the eye and the large size of the pupil modify the visual acuity by reason of circles of diffusion. The corrected vision of

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[1] Snellen, On the Methods of determining the Acuity of Vision. System of Diseases of the Eye, by Norris and Oliver, 1897, vol. ii. p. 23.

an ametropic eye is found to be inferior to the normal in the proportion of three to four per cent., without its being possible to demonstrate any other cause for this impairment than the dioptric condition of the organ. Even after correction, myopes of more than three diopters only enjoy the physiological visual acuity, $\frac{6}{6}$, in the proportion of about thirty per cent.; while above six diopters not more than three per cent. have such a degree of sight. This defective vision in strongly myopic eyes is improved when the patient is brought near to the chart, while under the same conditions in the hypermetrope it diminishes as a result of prolonged efforts at accommodation. It is well known that persons whose work demands constant near-vision have a degree of visual acuteness that is inferior to those who make use of distant vision. Moreover, the two most important subjective symptoms of astigmatism are diminution of visual acuteness and asthenopia. Knies has remarked with reason that a great number of those who feign amblyopia are cases of unrecognized astigmatism.

Schmidt-Rimpler[1] calls attention to a cause of amblyopia which is often unrecognized,—namely, the existence of very fine, almost transparent, infiltrations in the centre of the cornea, that are difficult to recognize if oblique illumination is not used with the greatest care, and which are the consequence of old and sometimes slight affections of the cornea or conjunctiva.

It is always well to give the subject a preliminary trial with the stenopæic opening, which should produce a notable diminution of visual acuity in an amblyope. After having varied in regular proportions the distances and the sizes of the test-types, the types should be arranged in such a way that letters of different sizes are read at the same distance. With this object, two scales can be arranged side by side on the wall, the first line of the one being continued on the second chart by letters of smaller size. If the patient, after having read the latter easily, declares himself unable to decipher the letters of larger size which compose the second line of the first scale, he betrays his imposture.

Bélow[2] and Fitow[3] advise showing the Snellen letters or symbols one by one, choosing characters of smaller dimensions than those which correspond to the distance of the patient. The distance which separates the malingerer from the test-types is then to be constantly diminished, but to a less degree than the size of the types.

Another class of patients, determined not to read the letters which they see, betray themselves by the movements of their lips, corresponding with

[1] Bemerkungen zur wirklicher und simulirter Sehschwäche und Gesichtsfeldeinengung. Festschrift zu ein hundertjährigen Stiftungsfeier des medizinisch-chirurgischen Friedrich-Wilhelms Instituts.

[2] Contribution à la détermination de l'acuité visuelle, chez les conscrits soupçonnes de simulation de l'amblyopie. Vestnik oftalmologii, 1889, March-April.

[3] De l'acuité visuelle dans les anomalies de la réfraction. Vestnik oftalmologii, 1888, p. 477.

the sound of the letter which they are on the point of pronouncing, by naming another, or, indeed, they designate a letter the shape of which is almost like it,—C instead of G, for example. It is also advantageous to compare the results of testing distant vision with those of reading at the near-point.

In case the malingerer does not pretend to have a pronounced defect of vision, recourse with much chance of success must be had to the trial with the mirror. For this purpose the patient is placed in front of and very near to a mirror in which a chart of reversed letters is reflected, so arranged that the image is formed at five metres from him. Not knowing this distance he will read the progressively smaller letters, and his visual acuity, if the case be one of simulation of amblyopia, will be thus determined.

Placed midway between a mirror and chart of test-letters or symbols at which he looks, the patient can be asked to name the smallest letters. After he has been made to turn half-way round, he is to be requested to read letters or signs of the same size, but which are not changed by reversal in the mirror, such as H and O. If he succeeds, he shows thus a visual acuity at least three times higher, since, on the one hand, he is placed at a distance from the image three times greater than in the first part of the experiment, and, on the other hand, the intensity of the illumination has diminished in an equivalent degree (A. and E. Barthélémy).[1]

Helmbold's method[2] is a variation of these tests. He uses two charts of characters, one with ordinary letters, and the other with the letters printed reversed; these being intended to be read by reflection from a mirror. This arrangement, in the writer's opinion, excites the suspicion of the patient, and it is of more value than the method to employ a chart of symbols.

The study of the field of vision,[3]—that is to say, of indirect vision,—of the color-sense, and of the light-sense in a malingerer will often lead to gross contradictions. So far as the field of vision is concerned, the most fantastic outlines, having no relation with pathological contractions or with physical and physiological laws, may be obtained. In addition, there is a valuable means for detecting fraud : that is, the campimetric and perimetric measurement of the limits of the visual field and of vision for white and other colors, *at different distances and on different dates.* In this experiment the patients who cannot remember the limits of the visual field in the different meridians often betray themselves by the tendency which they have to assert that they see at practically the same place on the perimeter or campimeter the test-object which is submitted to them, whatever may be the size

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[1] E. Barthélémy, Amblyopie double simulée procédé pour la déjouer et mesurer l'acuité visuelle. Archives de médecine et de pharmacie militaires, 1894, t. xxiii. p. 285.

[2] Ueber Simulation. Klinische Monatsblätter für Augenheilkunde, 1896, Band xxxiv. S. 217.

[3] K. Baas, Das Gesichtsfeldeinengung, 1896. H. Wilbrand, Perimetry and its Clinical Value. System of Diseases of the Eye, by Norris and Oliver, vol. ii. p. 313.

of the angle,—that is to say, whatever the distance which separates them from the apparatus may be. A certain number of malingerers imagine, moreover, that the farther the eye is situated from the fixation-point on the campimeter or perimeter, the less distinct should be the vision, and the narrower the limits of the field of vision.

As a general rule, malingerers are unacquainted with central and annular scotomata, hemianopic gaps, and only complain of very marked concentric contraction. It is necessary to attach great importance to their first assertions and to compare these with the subsequent ones.

As in the case of central visual acuity, the measurement of the peripheral vision presents difficulties and cannot be gotten to a mathematical certainty. It differs with the different instruments that are employed to determine it. Perimeters, for example, have not all the same degree of regularity of curvature, so that the distance from the eye to the fixation-point is even in that way made different. As for self-registering perimeters (Stevens and McHardy), they may furnish erroneous answers as a result of stretching which the cords pulling the test-disk in action undergo.

The position of the eye in the orbit, the carriage of the head, the conformation of the face, the width of the palpebral fissure, the diameter of the pupil, the depth of the anterior chamber, the size of the test-object, the state of the refraction and accommodation,[1] etc., all are of the greatest importance. According to the observations of Uschakoff, Reich, and Pietsch,[2] the visual field of the emmetrope is a little larger than that of the myope, and a little smaller than that of the hypermetrope, for white, blue, and red. Moreover, the visual field is increased by the act of accommodation, which increases the refractive power of the crystalline lens, and brings the pupillary plane nearer the cornea (Liebreich, Aubert).

As in the case of central visual acuity, the limits of the field of vision are normally somewhat variable in different subjects, and at times even in the two eyes. Thus, it is not rare to find contractions of ten degrees and even more in patients without there being a question of a pathological condition, while, in other subjects, the field of vision is found to be more extensive than that of the normal average. It is indispensable, therefore, to take into consideration these causes of error when the field of vision in a neurotic subject and an individual affected with hystero-traumatism is to be studied.

The examination of peripheral vision according to the methods of Bjerrum[3] or of Groenouw[4] gives appreciable results, and allows the obser-

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[1] Guillery, Accommodation und Gesichtsfeldeinengung. Archiv für Augenheilkunde, 1898, Heft 3, S. 272–300.

[2] Die Ausdehnung der Gesichtsfelder für weisse und farbige Objecte bei verschiedenen Refractionszuständen. Inaugural Dissertation, Breslau, 1896.

[3] Undersoegelesen af Synet, Copenhagen, 1894.

[4] Ueber die Sehschärfe der Netzhautperipherie und eine neue Untersuchungsmethode derselben. Archiv für Augenheilkunde, 1893, S. 85.

vation of alterations that escape recognition by the ordinary methods of examination. Bjerrum advises placing the patient before a black curtain, situated at two metres' distance, before which the observer, wearing black gloves, moves small ivory disks of different sizes that are attached to black sticks one metre long. Groenouw arrives at analogous results by making the patient fix upon a black point placed on a white ground.

Simulation of partial achromatopsia and of dyschromatopsia can be detected by means of Stilling's isochromatic tables, or, by preference, by means of those of Pflüger, which are based on the phenomenon of contrast. The color-blind subject who looks at gray letters that are printed on a purple sheet and are covered by tissue-paper, confuses them with the groundwork, which appears gray to him; while a normal eye perceives the letters which stand out by contrast in their complementary colors. In the same way the patient who declares that he confuses red and green if he is made to look at these two colors through a red glass ought to see the green darker than the red.

In the study of color-perception, the condition of general illumination, that of fatigue or rest of the retina, the intensity of the light, the purity of the colored surfaces, and of the background against which they appear, should all be taken into consideration. Langley[1] has established the fact that the color-sense is appreciably variable in the normal state.

As in the case of visual acuity, it is also necessary to distinguish between central and peripheral perception of colors. The latter is determined in a similar manner as peripheral visual acuity, except that the test-objects are replaced by colored tests, made in the order of green, red, and blue.

A considerable diminution of central visual acuity is habitually accompanied by the appearance of a central scotoma for colors. The malingerer who is ignorant of this relation will usually betray himself by his answers.

Certain malingerers, in whom the light-reflex is normal and who can count fingers easily at a certain distance, deny that they can perceive a candle-flame that is moved in their visual field at two metres' distance, although ophthalmoscopic examination may fail to show anything abnormal (Groenouw[2]).

In other instances tests with the photometer may be negative although before the sitting is over the subject may very easily find, for example, his spectacles that have been placed on a table in a dark room (Groenouw).

## III.—HYSTERO-TRAUMATIC AMAUROSIS AND AMBLYOPIA.

Particularly difficult sometimes is the diagnosis of hysterical or hystero-traumatic amaurosis and amblyopia. The study of the complex morbid state, which is found in a great number of the victims of serious injuries

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[1] Energy and Vision. American Journal of Science, 1888, pp. 359–379.

[2] Ueber einige Mittel zur Entlarvung simulirter Schwachsichtigkeit. Monatschrift für Unfallheilkunde, 1894, Nr. 6.

and in particular of railroad accidents, has been enriched in recent years by numerous works, which have thrown some light on the pathogeny of these manifestations, of which some at least are most fantastic, and were formerly ascribed to malingering. Formerly, when the hystero-traumatic neuroses were but little or not at all known, the medical expert, if he did not find some objective signs revealing an organic lesion in the complainant, came to the conclusion that the case was one of malingering. Now there are some exceptional cases in which objective symptoms are almost wanting, or which remain indefinite; but even in these types there remains a sufficient symptomatic grouping which is found uniformly in all such cases (including the injured who do not claim damages) for a competent observer to form his judgment upon. Moreover, the perfect simulation of a group of symptoms that are so complex will be most difficult even to a physician who is familiar with all the signs of the disease. It is doubtless unnecessary to deny simulation, but statistics show that it is much rarer than certain authors have been prone to assert.

If the hope of a large indemnity induces unscrupulous individuals to exaggerate or even to feign some subjective functional troubles, such as dazzling, intense photophobia, ocular or periorbital pains, inability to continue the work of reading or writing beyond a few minutes' time, etc., there are other symptoms, the simulation of which is difficult for a prolonged period. Such are the iris-reflexes, strabismus, muscular insufficiency, diminution of the visual field, lessening of the amplitude of accommodation, etc. Many subjects acquire nevertheless, by practice, the faculty of squinting outward, inward, and especially down and in; and we know that a certain degree of myosis is the result of the convergence. Schwarz[1] and Lechner[2] have published observations of patients who could voluntarily adduct and abduct one eye, the other remaining fixed on one point. Bechterew[3] and Szontagh,[4] have called attention to instances of voluntary dilatation of the pupil.

Exaggeration, which is common enough, is, on the contrary, usually to some extent of the unconscious and suggestive types. All medico-legal experts know that the delays and anxieties of a lawsuit aggravate most of the symptoms, while a verdict, as a rule, brings a sort of relief, with a notable improvement, even when the decision of the court is less favorable than might have been hoped.

The ocular symptoms of hysteria are, moreover, so peculiar that it is customary to describe them under the term of the "hysterical eye."[5] They

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[1] Ueber willkürliche einseitige Augenbewegungen. Centralblatt für praktische Augenheilkunde, 1897, August.

[2] Abnorme willkürliche Augenbewegungen. Archiv für Ophthalmolgie, 1898, SS. 596–613.

[3] Sur la dilatation voluntaire de la pupille. Nevrologiicheski vestnik, 1895, iii. 1.

[4] Dilatation voluntaire des pupilles. Le Mercredi médicale, 1896, p. 209.

[5] Pansier, Les manifestations oculaires de l'hystérie. Thèse de Montpellier, 1892.

consist essentially in an association of sensory disturbances and disorders of the intrinsic and the extrinsic muscular systems of the visual organs.

Amblyopia or amaurosis of the hystero-traumatic type, which is ordinarily unilateral at first, is characterized by the absence of ophthalmoscopic signs and clearly manifest anatomical lesions, by an abolition or a more or less marked lowering of central vision, a diminution of the light-sense, a concentric contraction of the field of vision for white and colors, and by an inverted order of the color-fields. Other more or less constant ocular symptoms (paresis of convergence) (Hubscher), accommodative asthenopia, spasm of the iris and ciliary muscles, monocular diplopia and polyopia (Parinaud), astigmatism, macropsia or micropsia, hemianopsia, erythropsia or hemierythropsia, ophthalmic migraine, contractions, and less frequently paralyses of the extrinsic muscles, inequality of the pupils, etc., can all be seen: while a characteristic evolution, variability, mobility, and contradiction of symptoms, abruptness of onset and recovery, the effect of suggestion, of the magnet, etc., are all recognizable.

Inequality of the pupils, considered until recently as a sign of organic disease, is observed in traumatic neurasthenia or hysteria, contrary to the opinion of Charcot, Bouveret, Seeligmüller, and others. The question that is usually given to decide is that of myosis and of spasmodic mydriasis of sympathetic origin. Nevertheless, Donath[1] and Fraenkel[2] have published a case of mydriasis which was due to paralysis of the common motor oculi (with abolition of action to light, and to accommodation, with insusceptibility to the action of eserine). This mydriasis habitually accompanies the sensitivo-sensorial forms of anæsthesias and amaurosis, but it is independent of the latter. It has the characteristics of an organic nuclear mydriasis, from which it differs by the spontaneous and the more or less rapid recovery at the time when the other hysterical phenomena disappear.

A symptom that is no less characteristic of unilateral hysterical amaurosis is its power of disappearing during binocular vision. Attention has not been given in this part of this work to the different interpretations that have been offered to this apparently paradoxical fact: the existence of a double cortical centre of vision (Pitres), or of a double function, of which one, special (binocular vision), may be intact, while the other (monocular vision) is affected (Parinaud); unconscious suppression of the normally perceived visual image by a dynamogenic action of the healthy eye that is excited by light (Féré, Bernheim, Janet), etc.

Amblyopia, at first unilateral, as the writer has said above, becomes bilateral at a more advanced period of the disease, and it is always more pronounced in the eye that is situated on the hemianæsthetic side. It is the same with the contraction of the visual field, which is regularly concen-

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[1] Hysterische Pupillen und Accommodations lähmung geheilt durch hypnotische Suggestion. Deutsche Zeitschrift für Nervenheilkunde, 1892, S. 217–290.

[2] L. Aurand and Fraenkel, Mydriase spasmodique et mydriase paralytique unilatérale hystérique. Revue de médecine, 1896, October 10.

tric, from the periphery to the centre, except in some cases in which there is at the same time a contraction and a modification in shape.[1] The limits of the field of vision for colors no longer correspond to the normal, nor do they extend to that of the field for white. Thus the limits for blue contract most, and pass within those of red. At the same time a reduction of the light-sense and of the color-sense is found. Violet and blue disappear first, followed by green and yellow, and finally by red. The field of vision, the light-sense, and the color-sense follow the variations in sensation, and present the phenomena of transferrence. It is altogether exceptionally that an enlargement of the field of vision has been observed with hyperæsthesia of the light-sense (S. L. Freund[2]) and the color-sense (Frinkl-Hochwart and Topolanski[3]). Asthenopic troubles, spasm, and paresis of accommodation are common and very obstinate, but they are essentially variable in their intensity. Most of the patients complain of migraine, a feeling of tension, pains in the eyes and around the orbit, photophobia, and dazzling. They are also greatly disturbed by the impossibility which they find, after a few moments of application, to continue to read or write on account of an increasing tendency of the headache and a doubling of the letters (nervous or hysterical asthenopia and kopiopia).

Very often, in addition, there are spasm of the accommodation and a slight degree of ptosis, with lacrymation, and a variable amount of injection of the conjunctiva.

Almost all the cases present hemianæsthesia with insensibility, and even analgesia of the cornea, the conjunctiva, and the eyelids. Disturbances of reflex, attacks of palpitation, fibrillar trembling of the muscles of the upper limbs, more particularly of the lips, with spasms, persistent insomnia, and gastro-intestinal symptoms (Bouveret), perversion of taste and smell, disturbances of speech, etc., may all be present. Hypnotism and suggestion exert a most remarkable influence. Borel has succeeded in inducing ptosis and strabismus by suggestion, just as Babinski has managed to produce an attack of ophthalmic migraine. With a certain amount of experience, amblyopia and most of the ocular disturbances just pointed out may be made to disappear by the same means. A great many authors, such as Fontan, Alario, Sgrosso, de Bono, Hitzig,[4] etc., have published conclusive instances of this ability.

The establishment of all these symptoms in an injured person of suspected neurotic heredity, and who fails to show any sign of constitutional disturbance (alcoholism and syphilis), permits the diagnosis of a hystero-traumatic amaurosis or amblyopia.

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[1] J. K. Mitchell and de Schweinitz, A Further Study of Hysterical Cases and their Fields of Vision. Journal of Nervous Diseases, 1894.

[2] Ueber cerebral bedingte optische Hyperästhesie. Neurologisches Centralblatt, 1892, Nr. 17.

[3] Zur Kenntniss der Augensymptome bei Neurosen. Beiträge zur Augenheilkunde, 1893, Heft ii. S. 46.

[4] Ueber einen durch Strabismus und andere Augensymptome ausgezeichneten Fall von Hysterie. Berliner klinische Wochenschrift, 1897, Nr. 7.

The facts, however, that the patient has no appreciable lesion of the eye, that he retains his normal iris-reflexes, and that, although blind in monocular vision, he nevertheless enjoys binocular vision, and moves about without hesitation in a neighborhood which is unknown to him, especially when he thinks he is unobserved, give suspicion that there is imposture. This is particularly so if, when subjected to various experiments, he falls into the ordinary mistakes of malingerers. Undoubtedly, hysterical subjects may feign amaurosis, but it is more frequently a question with them of an involuntary simulation. Moreover, it is sometimes difficult to decide without any reservation, unless the patient confesses, between a simulated and a hystero-traumatic amaurosis or amblyopia. In the latter case it is true that most frequently other visual and general symptoms of a special type that are peculiar to the nervous affection which make the diagnosis clear are associated with it.

The question arises, is it necessary to consider the concentric contraction of the field of vision in patients that are affected with hystero-traumatism as always imaginary and feigned, as certain authors contend (Hirschberg,[1] Nieden, *loco citato*), for the reason that with these individuals orientation is never affected, while it is markedly disturbed in cases in which, for instance, there is a contraction that is symptomatic of an organic affection of the visual apparatus, as atrophy of the optic nerve? It may be remarked, with Groenouw, that in the street such a patient, by relaxing his accommodation, possesses a larger visual field than that which has been measured on the perimeter, and that the periphery of the retina, which is insensible to small test-objects, is not so to the large ones that serve for ordinary orientation. In reality the images are perceived in an unconscious way over the whole extent of the retina, and the patient sees without taking any note of the fact. Concentric contraction of the field of vision, the pathognomonic mark of hysteria, as has been demonstrated (l'École de la Salpêtrière), and which is observed also in other ocular or general affections (anæsthesia of retina, hemeralopia, paralysis of the accommodation, traumatic neurosis, diphtheritic paralysis (König), etc.), is associated most frequently with another symptom, which consists in an instability of the limits of the visual field and a more marked contraction of them, according as the test-object is moved from the periphery to the centre of the perimeter or in the reverse direction. Most authors consider this symptom the result of a kind of fatigue of the nervous system which is proper to this category of patients, while others refer it to the visual centre (Schiele), to the retina (Wilbrand, O. König), or simply to physiological fluctuations (fatigue) of the attention. The believers in the last theory (Simon,[2] Salomonsohn,[3]

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[1] Eleventh International Congress of the Medical Sciences, 1894.

[2] Ueber die Entstehung der sogenannten Ermüdungserscheinungen des Gesichtsfeldes. Archiv für Ophthalmologie, Band xl., Abth. iv., S. 276–307.

[3] Ueber die sogenannte pathologische Netzhautermüdung, Berliner Klinik, 1894, Heft 174.

Fr. Schultze,[1] Voges, Schmidt-Rimpler[2]) have remarked that, far from always diminishing as a result of perimetric measurements continued for a long period of time, the field of vision enlarges with certain patients during the course of the examination, and that for this purpose it is sufficient to strongly attract their attention. The observations of Peters,[3] Karl Voges,[4] and Schmidt-Rimpler in the clinic at Göttingen have established the fact that the fatigue of the visual field, its instability, and the concentric contraction, pointed out as symptoms that are peculiar to the traumatic neurosis, are found equally in a large number of healthy subjects, and with greater reason in hysterical subjects. Hence arise these extraordinary visual fields, the limits of which vary, as it were, with the will of the physician who examines and arouses to a greater or less degree the attention of the patient. The variations in observation are produced from different causes, among which are differences in methods of examination and in the apparatus. It is necessary also to take into consideration the influence of the accommodation (Groenouw), the size and the illumination of the test-objects, the state of dilatation of the pupil, and the adaptation of the retina (Wilbrand).

According to Jolly,[5] untruthfulness is common among hysterical children, but in accordance with Schnabel,[6] it is not often seen. Malingering is possible with patients who are affected with traumatic hysteria, and is far from being rare, as Oppenheim and Knopp contend, but its existence must not be slightingly admitted. It is often a question, as the writer has previously said, of defective attention or of the psychical exclusion of the periphery of the field of vision, the proof of this being that if, unknown to the patient, this peripheral zone can be excited, the fact that it is sensitive becomes established.

Concentric contraction of the field of vision, in traumatic hysteria, has a much greater objective value in proportion as it is slight and conforms to physical laws (Wilbrand and Saenger[7]). Under such conditions it is extremely difficult to simulate. To expose this simulation recourse can be had to perimetric measurement, made at different and considerable distances, with test-objects of different sizes for white and colors. Another

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[1] Weiteres über Nervenerkrankungen nach Trauma. Deutsche Zeitschrift für Nervenheilkunde, Band i. S. 445.

[2] Ueber Gesichtsfeldermüdung und Gesichtsfeldeinengung mit Berücksichtigung der Simulation. Wiener medizinische Wochenschrift, 1895, Nr. 43.

[3] Ueber das Verkommen und die Bedeutung des sogenannten Verchiebungstypus des Gesichtsfeldes. Deutsche Zeitschrift für Nervenheilkunde, Band v. S. 302.

[4] Die Ermüdung des Gesichtsfeldes, neue Versuche, etc. Preisschrift und Dissertation, Göttingen, 1895.

[5] Ueber Hysterie der Kinder. Berliner klinische Wochenschrift, 1892, Nr. 34.

[6] Ueber eine Sehstörung durch Suggestion bei Kindern. Prager medicinische Wochenschrift, 1893, Nr. 10.

[7] Wilbrand-Saenger, Ueber Sehstörungen bei functionellen Nervenleiden, 1892. Wilbrand, Ueber die Veränderungen des Gesichtsfeldes bei den traumatischen Neurosen. Deutsche medicinische Wochenschrift, 1892, Nr. 17.

method for the detection of this simulation has been pointed out by Schmidt-Rimpler, and consists in an examination that is made with a thirty-degree prism.

Schmidt-Rimpler (*loco citato*) advises, in the latter case, that while the test-object is situated on the limit of the field of vision of one eye, but is still seen by the patient, a prism that is too strong to be overcome be placed before this eye, both eyes being open. It is necessary to make sure, before the adjustment of the prism, whether the test-object is seen by both eyes or by one only. In the former case, if there had been simulation, the patient would have said that he saw two objects, the true and the false image. If, on the contrary, he had been honest, he would have seen but one object. If, originally, the object is seen by one eye only, then, if the patient is truthful, the prism placed before this eye makes the object disappear; the patient sees nothing. In the case of simulation, the object remains visible, but with prismatically colored edges.

## IV.—FEIGNED BLINDNESS OF BOTH EYES.

Simulation of complete blindness is altogether exceptional, because of the extreme inconvenience which it entails in the acts of ordinary life. It would be necessary, besides, for the impostor to possess an exceptional degree of cleverness to be able to adapt himself suddenly to the classical attitude and gait of a blind man, who moves forward "all in one piece," with hesitating step, his hands stretched out before him, an expressionless face and dull stare, his eyes raised towards heaven, and eyelids that are motionless in front of a bright light, or before an object with which he is threatened to be struck. If objective examination, followed by methodically performed functional examination, has failed to reveal anything abnormal, and if the patient has an interest in deceiving, doubt at once arises. However, hysterical subjects, simple conscripts, or unfortunate amblyopes are occasionally seen who attempt to play this comedy.

The indications furnished by the direction of the visual axes, by the pupillary aperture, and the play of the irides are the most important. If both pupils remain dilated and the irides are motionless on a sudden access of a flood of light, blindness is probable. In order to obtain more complete information, the following question must be put to the patient: When did the absolute loss of sight occur? If he answers that he has been blind for a long time, this fact is a matter of common knowledge in his neighborhood, and ophthalmoscopic lesions, as a rule, should be found. If, on the contrary, he asserts that the blindness is of recent date, and if its onset has been sudden, it may, exceptionally, not be revealed by any actual objective sign. In this case time must be asked for before an opinion is formed. A close watch must be kept, and if it is a question of a lesion of general nutrition or of the nervous system, the diagnosis will be surely established some months later.

Schmidt-Rimpler[1] has recommended in these cases a test which consists in making the patient look at his own hand held at a short distance from the eyes. While the genuine blind man will succeed easily enough, because his muscular sense tells him the position of his hand, the malingerer, on the contrary, will affect to look in an entirely different direction, for fear of exciting the idea that he really sees. It is true, however, that, for the same reason, some who are really blind may behave in a similar way.

The simulation of double amblyopia is also difficult to expose, and in many cases it will be found impossible to determine with certainty the degree of lowering of visual acuity, since the indications by which a comparison of the normal organ with the one that is declared diseased are lacking.

Two types may present themselves:

1. The complainant declares that he can easily get about and recognize large objects, yet, when placed in front of a printed test-chart and very near the letters, he cannot distinguish them.

Having ascertained that the patient is an ametrope, that no ophthalmoscopic lesion is present, and that the field of vision and the color-sense are normal, he is asked to decipher some printed characters after his eyes have been provided with very strong convex lenses. If he does not admit any improvement, it is right to believe that he is voluntarily deceiving.

2. The patient claims only a diminution of visual acuity, and is apparently only able to read the large sizes of the test-type. In this type of complaint it is easier to take him by surprise. One or another of the tests which have been previously noted can be used, and in most instances will be decisive.

Though the simulation of binocular blindness involves so many difficulties and inconveniences that it has rarely been resorted to by expert malingerers, yet it has been quite frequently observed in hysterical subjects. All authorities have hitherto admitted that no objective test could be applied to its detection, and that only patient waiting and careful watching could discover it. Etherization has been suggested by Hutchinson, in the expectation that as the effect of the anæsthetic passed off the simulator would probably expose his ability to see before his consciousness was sufficiently restored to enable him to resume the deception.

Priestley Smith has recently proposed a valuable objective test which is based on the direction of the visual axis, as shown by the von Graefe prism test. This means of detection has long been known and occasionally used in the examination of cases of simulated monocular blindness. In 1867, von Welz suggested the placing of a prism of from ten to twenty-five degrees before the supposed amaurotic eye, with its base out, while the simulator is made to read. He states, "To see singly the eye will deviate inwards, and will recover itself the moment the prism is removed."

[1] Notiz für die Untersuchung auf Simulation von Blindheit. Berliner klinische Wochenschrift, 1876, S. 525.

It does not seem to have occurred to any one to apply this simple test to the detection of the simulation of binocular blindness until Priestley Smith employed it in the following case:[1]

"A few months ago a prisoner, awaiting trial for burglary with violence, awoke one morning to find himself, he said, blind in both eyes. The prison-surgeon had no doubt that he was shamming, but wanted positive evidence: so we examined him together. The man declared himself to be quite blind in both eyes, and acted the part of a blind man fairly well, though somewhat overdoing it. The irides were already under the influence of atropine, and could therefore give no evidences to light-reflex. A lighted candle was placed before him in a dark room. He was not required to look at the candle, being nominally blind, but the candle was placed about where he appeared to be looking. A prism, with its base inward, was then placed before one eye. Instantly the eye moved outward. The prism was removed and the eye moved inward. The man was told that his blindness would certainly disappear as quickly as it came, and he probably understood that the fraud would get him into more trouble if continued. His sight was soon restored. If this subject could have carried his blindness into the dock, a merciful judge and jury might not improbably have felt that a higher tribunal had already visited him with a heavy punishment, or at least that he was incapacitated for further crime, and would have dealt with him very leniently. As a matter of fact, he was a particularly daring and dangerous criminal, and had, during a previous imprisonment, attempted the life of a prison-surgeon. He received a long sentence."

In the performance of these tests a prism of ten degrees is a convenient one, as few subjects with binocular vision will be met with who will not unconsciously overcome its effects when it is held with its base towards the temple. The possibility of heterophoria must, however, be borne in mind, and the prism must be varied in strength and direction.

It is not necessary to darken the room and to use a flame. Indeed, it is perhaps better not to do so as an experienced malingerer might purposely avoid looking in the direction of the light. If a prism be worn for awhile in a lighted room, the eye will deviate to correct the diplopia of surrounding objects, and will resume its normal position when the prism is removed. Even if one eye is really blind, the seeing eye will move behind the prism; but, in that case, the blind eye will move in the same direction and to the same extent (Jackson). This form of movement may also occur if there is vision in each eye, but not if there is binocular fusional vision. In concomitant squint, in which the power of conjugate movement is not lost but binocular vision is, the squinting eye will move in the same direction as the other. When from a high degree of amblyopia, or other cause, the subject does not fix, the test may fail. In high myopia the test is

[1] British Medical Journal, June 20, 1896.

likely to fail for distant vision but may hold good for near. A malingerer might refuse to look in the direction of a flame, but would find it difficult, if he had good binocular vision, to avoid correcting the diplopia of surrounding objects in a lighted room. Although, therefore, the failure of either eye to move behind the prism is not an absolute proof of complete blindness, yet such movements make the presence of vision more certain.

# THE OCULAR SIGNS OF DEATH.

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In asking for an article upon the subject of the ocular signs of death the editors of this System have obeyed a very commendable impulse. They have aimed to satisfy the curiosity of those who ask if the organ which they have made their special study can play a useful part in the elucidation of the interesting problem of the signs of death.

To fulfil what is expected of me, I deem it necessary, without losing sight of my subject, to leave it for a moment, and to remind the reader what investigations have heretofore interested men concerning death, what motives have actuated them to study it scientifically, and what means have been employed for that end. As for the moral side, by which we mean the passing away of those with whom we have lived, their death imposes on us a duty,—cruel, it is true, but more imperative than cruel,—that of disposing of their remains. This end can be obtained only by the destruction of the body,—rapid, if it be consigned to the flames or if it be exposed to the action of very powerful agents; slow, if it be left to the disintegrating power of the earth. Before this is done, care should be taken to understand the signs which our bodies present after death,—signs that are capable of establishing the reality of death beyond peradventure. Because death is so complex an affair, it ought to be considered as the sum of a number of special deaths. To understand death it is necessary to understand life, and we know that our ideas of the one have kept pace with our ideas of the other. This problem has always remained uncertain and undecided.

Even in olden times evidences of research as to the signs of death are not wanting. Galen, Celsus, Zacchias, Ambroise Paré, and Haller have all left accounts of methods for the prevention of premature burial by the judicious observation of certain signs furnished by the different organs. Not

till the time of Louis, however, do we find the verity of the signs of death established, and the signs themselves and their significance systematically arranged. The celebrated work of Bichat did much to effect this purpose.

Little by little another branch of medical science developed whose influence made itself usefully felt. With the progress of civilization public hygiene became established, which not only dealt with the disposal of dead bodies and legal medicine, but sought to discover the secrets of the process of transition from life to death. Both of these subjects, with their precise questions and exact methods, encouraged precision and well-directed criticism in investigation. All this was not without value; for side by side with the good impulse given to the efforts of investigation appeared the baneful influence of public opinion. This, aroused by more or less apocryphal recitals of awakening in the coffin, which were exaggerated and strengthened by supposed scientific accounts taken from the transactions of learned societies, loudly demanded the discovery of signs that would be capable of enlightening professional men and of furnishing to legislators the right to enact measures for greater security. Some well-meaning persons thought to solve the problem by offering prizes which were to be awarded by the academies. These, unfortunately, reserved the right to fix the conditions, not suspecting that to propose questions concerning scientific matters it was necessary to possess an instinct which was not theirs, or a knowledge which had not then been acquired. Thus, the Marquis d'Ourches, about 1872, made the following bequest:

"I desire that there may be deducted from my estate the sum of twenty-five thousand francs, intended, under the conditions hereinafter named, for the founding of two prizes,—to wit:

"1. A prize of twenty thousand francs for the discovery of a simple and easy method of recognizing, in an unequivocal manner, the signs of real death, the principal condition of this prize being that it shall be practicable to poor and uninstructed villagers.

"2. A prize of five thousand francs for the discovery of a method of recognizing, in an unequivocal manner, the signs of real death by the aid of electricity, galvanism, or any other procedure requiring either the intervention of a man of arts or the application of knowledge, the use of instruments, or the employment of substances which are not within the reach of everybody."

So tempting an offer could not go unanswered, and one hundred and two candidates presented their work to the Academy. As a matter of fact, the Academy did not award the prize of twenty thousand francs, and divided that of five thousand francs. Careful study of these methods, however, compels us to separate the truth from a mass of opinions which could not be verified by their authors.

The ocular signs of death are not many. Those of the adnexa of the eye are of but little value. The conditions of the eyebrows and the lacrymal apparatus have not much to teach us. They participate in the general

relaxation which appears in all the tissues at the moment of the last breath, and they play their part in giving to the face the appearance of repose which succeeds the storm of the death-agony.

"The lids are half open," said Tourdes, "and this is natural, since they are elevated by the simple tonicity of the frontalis muscle when the contraction of the orbicularis no longer opposes it." This, as the learned professor of legal medicine at Nancy says, is dependent upon paralysis of the seventh pair, which precedes that of the third. There are, however, some exceptions to this rule of position, and if any value is attached to it in death from disease, it loses this value in cases of violent death. During the war of 1870 how many dead bodies could be seen with their eyes wide open! Again, in contradistinction could be cited an accident on the Seine, where twenty-eight persons, of all ages and both sexes, were drowned on a hot July day. Here, three or four hours after death, in most of the bodies, the lids were puffed and closed by a semi-œdematous swelling. So that, taking into account popular opinion, which recognizes the former fact in the well-known expression, to "close the eyes of the dead," and remembering what Camper says, "No one dies with mouth and eyes open," we are obliged to believe that so many causes can modify the position of the lids during death that it is impossible to make a certain sign of it, thus rendering it necessary to look with suspicion upon all argument that is advanced in this connection. Again, cases may occur where the organic muscles of the lids become sunken, applying the lids to the globe, and making between it and the edge of the orbit the depression which gives to the face of the dead something of its peculiarity.

Retraction of the globe in the period of cadaveric rigidity constitutes a much later sign. Its manifestation, however, is so uncertain of demonstration that no one has dreamed of giving to it the least importance. Bouchut, in his account of the death-agony, has described a sudden retraction of the eye. He says that the organ appears to become sunken, resuming its place at the very moment of death. It is evident that here a spasm which is one of the last expressions of life has the same effect as the cadaveric contraction that appears later.

Between the half-open lids the globes of the eyes are seen, whose "optic axes have become parallel," said Tourdes, and he adds that "often parallelism is exceeded, the eyes having a tendency to divergent strabismus." Müller, in a thesis at Strasburg in 1870, describes a slight rotation of the eyes from below upward, which, as the body rests upon its back, causes the eyes to appear to look at an object situated behind and over the head. In this we recognize the result of a non-simultaneous paralysis of the nerves which supply the ocular muscles. This position of the eyes, however, can be disturbed by different circumstances, and should not be regarded as characteristic of death.

Insensibility of the conjunctiva and the cornea, like general anæsthesia, is more striking here than is elsewhere seen. This is so on account of the

exquisite sensibility which these portions of the visual organ exhibit during life. Tourdes tells us that sensibility does not disappear simultaneously in both cornea and conjunctiva. The former membrane, which is supplied by filaments from the ophthalmic ganglion, retains its sensibility longer than the latter, which is supplied by filaments from the fifth pair. Furthermore, sensibility passes away differently according to the kind of death. Section of the globe, etherization, poisoning by curare, and strangulation paralyze the conjunctiva before they do the cornea; while cold and strychnine kill the cornea first, and leave the conjunctiva, especially towards the inner angle, longer sensitive.

Absolute insensibility of the two membranes does not prove death, it being often seen whilst life undoubtedly exists,—as, for example, during anæsthesia and syncope. Moreover, how frequently has it been observed that the condition can be determined only by subjective phenomena, which must be manifested by patients who are conscious and able to express their feelings!

The color and the appearance of the vessels on and under the conjunctiva should have great value in this connection, since they are readily observed objective signs. Exsanguinated vessels can be easily distinguished from full ones, and the color of living tissues is readily differentiated from cadaveric lividity. The character of death and the degree of the surrounding temperature have very marked effects on the signs. In an emaciated subject, dead of disease for a moderate length of time, in winter, the pallor of the conjunctiva is characteristic. In a person dying suddenly in warm weather a lividity of the same tissues, accompanied by slight œdema, is often present. Certain hemorrhagic phenomena are usually more the signs of the mode of death than they are of death itself.

In a collection of theses by Haller, there is one by Camper upon the cause of the dimness of the cornea in dying persons. It is dated 1746. In it is the following expression: "Constans est observatio, morientium oculos suum amittere splendorem." In 1752, Louis wrote, "The cornea of the dead is usually covered with a very delicate glairy film, which breaks into many pieces when any one touches it, and which may be easily removed by wiping the membrane. This film makes the cornea somewhat dim, and may obscure the deeper structures."

Danois, in a memoir printed in 1721, stated that he had been diligent in seeking the cause of this deposit. He finally came to the conclusion that the film exuded through the cornea. Verdier and Camper were of the same opinion. In France a popular saying, when this deposit forms on the eyes of the dying, is, "See what has happened; the eye has burst!" At Metz they say, "There is no more hope; the tear-sacs have burst!" At Lyons the remark is that the dying "have a veil before their eyes."

An explanation given by Winslow, while very interesting, is incomplete and based upon incorrect anatomical ideas. He says that by pressing upon the eye he could make a liquid exude through the cornea. Unfortu-

nately, he had not discovered what we now know so well, since the work of Leber and Schwalbe on the circulation of lymph through the ocular tissues, that the intra-ocular fluids exude through the cornea without pressure. In 1849, more than one hundred years after Winslow's observations, Bouchut, still believing that pressure was necessary to the formation of this film, asserted that this condition was caused by spasm of the recti muscles. It is well known now that pressure plays a very small part in the process, and that osmosis is sufficient to carry the necessary moisture to the corneal surface.

Recent anatomical researches have shown that the superficial layer of the cornea is composed of stratified epithelium. The necessity of maintaining a high polish on the corneal membrane by the action of the lids requires that the epithelial layer shall be kept moist. Hence, in the normal state, there is a smoothness and a fragility of this superficial tissue which is in a state of constant reproduction. The brittleness is so great that, if a cornea be examined with sufficient magnifying power and good illumination, abrasions, appearing either as irregularities in the surface of the membrane, which are best seen by reflected light, or as local opacities, may often be found. If this normal play of functions is interrupted, and the lids cease to sweep the eyes, the polish quickly disappears, and the epithelium gradually dries and hardens. If the intra-ocular fluids are protected from evaporation, the membrane swells and softens. Sometimes both of these effects manifest themselves in the same cornea, especially if it be partially protected by the lids. It is apparent, however, that the formation of this film, which Louis so well described, may occur during life, and that hence its value as a sign of death is limited. Moreover, it must be remembered that in the dead the condition always appears without any complication. In the living, as a rule, it accompanies reactionary phenomena, with which generally are associated the products of inflammation and glandular secretion, such as pus and mucus. That this is not invariably the case is shown by the fact that it is found in grave fevers such as typhoid, in meningitis, in cerebral affections, and in diseases characterized by coma and syncope. Finally, the corneal deposit is not produced at any definite time: thus, even twenty-four hours after death the eye may be transparent. Especially is this the case in subjects who have succumbed to a very brief illness, in which the organism has suffered but little.

Dependent upon the transudation of the ocular fluids through the cornea, a shrinking of the globe first manifests itself on that membrane, forming folds which Bouchut has described as capable of obscuring the underlying parts. Later the folds blend themselves into a sort of depression, as may be seen in some cataract patients, where, after the corneal section has been made, the aqueous humor escapes. "The eyes of the dead," says Louis, "become flabby and soft in a few hours. There is no disease, no process in the living human body, which can produce such a change. This sign is truly characteristic, and I give it as indubitable.

As long as the globe of the eye preserves its natural hardness, no one can say that the person is dead, whatever be the other signs that indicate it. The sinking in and softening of the eyes warn us to expect putrefaction." A statement so emphatic, and emanating from such high authority, deserves attention. Bouchut writes, "As for the softness and sinking in of the globe, this double phenomenon occurs at the instant of death." As this phenomenon has nothing to do with loss of ocular tension, but depends upon flaccidity from exosmosis of the contained fluids, it is probable that this authority is mistaken in his appreciation of the sign. Josat strongly opposes the value of the sign, and says that the authority of Louis has given credence to a phenomenon which, to his personal knowledge, has at times failed to mean death. "Often, also," he says, "the patient has lived more than eight days from the moment softening of the globe could be demonstrated." He further believes that just previous to death in certain cases the eyes, like the rest of the organs, tend to return to their former condition. Orfila does not attach much value to softening of the eye as a sign of death. He says, "We know that asphyxiated people whose eyes were flaccid have been restored to life, and that others whose eyes were softened have had the eyes resume their hardness after death," etc. Devergie, who was intrusted with the reports for which the Academy of Medicine was indebted to the bequest of the Marquis d'Ourches, states, "As for the eye in certain grave fevers, in epidemic malignant small-pox, and in cholera, not only may the film on the cornea form before death, but the cornea may become wrinkled during life, as it does in death." In the discussion which followed the reading of this report, Clin said that if the cornea became flaccid or wrinkled it was because the fluids escaped through the ocular coats, to spread out on the lids or to evaporate on contact with the air, the condition thus showing itself to be a simple matter of transudation. He also said that if the eye be opened at this time it will not emit any marked putrid odor. Further he stated that it is true, therefore, that the eye becomes flaccid and wrinkled by reason of the fluids, and these purely physical phenomena are the same in life as they are in death: in life many physiological conditions exist solely to protect the fluids from transudation: if general death or profound morbid disturbance disarranges these conditions, evaporation takes place, and this sign manifests itself. It still remains, then, to find a single phenomenon—a factor in the great product which we call death—that is pathognomonic of the condition.

In a communication made to the Academy of Sciences, Larcher refers to discoloration of the sclerotic as a true sign of death. This sign, however, had been pointed out in 1833 by Sommer, who supposed that after death the eye dried in the air, allowing the sclerotic to become transparent and to permit the chorioidal pigment to be seen, thus producing the discoloration. In Larcher's opinion, this discoloration is produced by an imbibition of the pigment by the sclerotic, and consequently is a product

of putrefaction. The phenomenon is characterized by the presence of a blackish discoloration, which, as a rule, develops first on the portion of the sclerotic to the outer side of the cornea that is not covered by the lids. Following this, another less black area appears on the inner side. Still later these two areas extend laterally, approach each other, and finally unite to form a part of an ellipse with its concavity situated downward. In a few instances Larcher has seen the sclerotic discoloration begin at the inner side. Sometimes a lividity of the skin precedes this discoloration of the eye. More frequently it manifests itself at the same time, and still more often it is not noticeable until much later.

It appears more rapidly in a warm atmosphere. It is more frequently found in children, in phthisical patients, and in those who have died of typhoid fever. What, we may inquire, is the nature of this sclerotic discoloration? Is it the result of a rupture in the tissues dependent upon simple pressure of the blood circulating in the capillary vessels, or is it a phenomenon of cadaveric imbibition? The latter supposition is the more probable. In fact, the dark spot on the sclerotic is the stigma of death, and, as it were, the advance-guard of putrefaction.

In 1873, Gubler pointed out that sclerotic discoloration is often observed in cholera patients some hours before death. Duchaussoy distinguishes two kinds of spots in such eyes,—one a true subconjunctival ecchymosis, and the other the visibility of the chorioidal pigment, which has been produced by a desiccation of the sclera. A remarkable fact, however, is that by restoring the natural moisture to the fibrous coat by injections of water the dark spots can be made to disappear, thus showing that they have been produced by drying.

Gayat, in 1876, informs us that the sclerotic discoloration may be seen in cholera cases, often in phthisical patients, and in maniacs who will not take nourishment, thus making a longer list of subjects upon whom the condition can be demonstrated before death. This is also the opinion of Blaier, who has often seen it during the death-agony of cholera patients.

Devergie holds that a distinction must be made between a spot arising from desiccation and one that has been produced by pigmentary imbibition. The writer believes that this difference has not been established, though he leans very strongly to the desiccation theory. This he does upon account of the position of the spots, their progress, their relation to the uncovered portion of the ball, their appearance, and their development at the late stage of certain wasting diseases; finally, he is influenced particularly by the possibility of the spot being made to disappear, in some cholera patients, by injections of water. It is, then, to his mind, a sign that should be classed with shrinking of the cornea and globe. It has the same origin and progress, and consequently the same value. Further, to him it is proof that the eye may appear to die before the rest of the organism, and also seems to revive with it: it cannot, therefore, be considered a certain sign of death.

Leaving for the present the tissues to which the above observations have been confined, and considering the deeper structures of the eye, the condition of the pupil and the fundus of the eye should be sought for before the dimness of the cornea prevents a proper view of these structures.

Bouchut, in 1849, drew attention to a condition of dilatation of the pupil at the moment of death. He did not deny that the phenomenon had been long known and described by old and modern physiologists, but to none of them did he attribute a knowledge of its value as an immediate sign of death, and especially as an early and positive symptom. Galen, whom he quotes, has said, "dilatur (pupilla) etiam in ipsa morte." Later, Whytt demonstrated on cats that had been killed by gunshot wounds that an enormous pupillary dilatation appeared simultaneously with cessation of the heart-sounds. Bichat has declared that the iris-movements cease at the moment of death, and cannot be recalled by any means a few moments later. In citing the opinions of this anatomist, Bouchut asserts that this is "a doubly incorrect statement." Evidently he has made himself the champion of the value of pupillary dilatation. He described it first, and assigned its cause to the relaxation of the sphincter pupillæ; he noted its appearance either a little before death or immediately after, compared it to pupillary contraction in sleep or in the death-agony, and finally made use of it not only to characterize death, but even to distinguish the condition from somnambulism. He further demonstrated to his own satisfaction that atropine and other substances do not affect the pupil of the dead subject. He said, "You believe that a pupillary dilatation takes place at the moment of death. This is not always so, for see what happens in chronic meningitis and in marked hydrocephalus. Here, before, during, and after death, the slightest mobility in the iris cannot be discovered. You tell us that at the moment of death a marked dilatation of the pupil occurs. It may be possible, though the power has not yet been granted to us to recognize that supreme moment, in spite of all that we have done to effect that purpose."

In 1862, Larcher does not even mention the pupil as the seat of any important sign of death. Is this silence attributable to a spirit of controversy? It is impossible to believe that a physician boasting of having filled for twenty years the position of verifier of death in Paris could overlook so important a sign, when his attention must have been attracted to it by the rewarded work of his opponent.

Tourdes, in his article "Cadaver" in the "Dictionnaire Encyclopédique des Sciences Médicales," confirms the observations of Bouchut. He declares that he has seen the pupillary dilatation in a woman who died from rupture of an aneurism of the aorta. "This dilatation," he adds, "lasted only a certain time; then, when rigidity set in, the pupil began to contract." As examples of these modifications in this case, he cites, "four hours after death the diameters of the pupils were seven millimetres; twenty-four hours after, four millimetres in the right and three and one-half in the

left; at the end of forty-eight hours they were two and three millimetres; after four days, three and three and one-half. The fifth day the pupils were contracted,—a difference in a few hours of perhaps three or four millimetres. For four or five hours after death the iris preserved its contractility under the influence of a galvanic current. It reacted also, but for a much shorter time, to instillations of atropine and eserine."

In spite of all these details, Devergie, in his report to the Academy of Medicine during the competition for the prize of the Marquis d'Ourches, does not even mention pupillary phenomena as ocular signs of death. Gayat, in a memoir on this subject, gives the following observations made upon a number of decapitated persons. In one, observed an hour and a half after execution, the pupil was moderately dilated, and an instillation of atropine was devoid of effect. In a second case, observed after the same period of time, the pupils were moderately dilated, and atropine failed to exert any influence on them. In a third instance, observed after three hours and a half, the pupils were moderately contracted. Finally, in a patient who died of phthisis, a pupil of three millimetres was observed at the end of one hour.

To help answer this question by personal experiences, I observed the following conditions in two rabbits that were killed in my laboratory in order to study the post-mortem effects of urinary poisoning:

The first animal died following an injection of ten cubic centimetres of urine. Myosis appeared at the moment of death. At the end of eight minutes this condition commenced to pass away. The pupil assumed an oval form, with its long axis vertical. In thirty-five minutes the pupils became round and slightly contracted. At the end of three-quarters of an hour after death the pupil resumed its normal size, and from this time it dilated. Full dilatation occurred at the end of three hours.

The second rabbit died after an intravenous injection of five cubic centimetres of urine. After myosis, the pupil began to dilate until five minutes after death, when it took an oval form, with its long axis vertical. This it maintained for ten or fifteen minutes. Twenty-five minutes after death it returned to its normal size, and continued to dilate slowly. The dilatation was marked at the end of two hours.

From this we must conclude that we are far from seeing the picture which Bouchut drew of the pupillary movements before, during, and after death, and that we cannot accept it at the valuation which he has given to it. May we not be nearer the truth in thinking, with Bichat, that the size of the pupil remains what it was at the moment of death? Again, what are we to say of the uncertain value of the supposed negative influence of mydriatic and myotic agents, should we agree with the findings of the experiment of Gayat upon a decapitated rabbit? In all cases, the employment of belladonna being very common in therapeutics, it is important, before examining the pupils of a dead person, to inquire if during

his last hours a mydriatic had been administered in any form. Further, there are so many ocular affections which prevent the pupil from dilating that these must be taken into consideration. Moreover, Gayat has properly observed that the movements of the iris and the size of the pupil may depend upon the projection of the lens forward, and upon the degree of effort which it makes to pass through the pupil. This projection, he says, may be favored by exosmosis of liquids from the anterior chamber, and in this way the pupillary sign become useless. Finally, Gunnhagen, of Königsberg, has studied the influence of temperature upon the movements of the iris in mammalia after death. He observed that at the temperature of the blood the pupil dilates enormously in the enucleated eye of a cat. He also found that it contracted again as the temperature was lowered, to dilate once more when the specimen was at zero. Whatever may be the interpretation given to these findings, the temperature must always be taken into consideration before attributing any value whatever to pupillary movements. If we are so conservative about the sign extolled by Bouchut, how much more so should we be in regard to the procedure of Ripenelt, of Dijon, who asserted that he had found a certain sign of death in the transverse shape of the pupil produced by pressure and counter-pressure upon the globe! Josat, who also noticed it, expresses his opinion of it in these words: "1st. The phenomenon in question can be obtained several hours before death occurs. 2d. It is often equivocal in its results. 3d. It may, as has happened, occur in a person attacked with cataract. In a word, the method is neither certain nor practicable in all cases, and ought to be rejected." To sum up, when Bouchut wrote, with Griéroult, that the pupil is the window of the soul, he employed an expression which is more poetical than true, which we should avoid repeating, and upon which it is needless to lay any stress. In 1876, Almès advised a procedure for the determination of death, which consisted in puncturing the cornea and evacuating the aqueous humor. He says, "If the pupil contracts, life still exists; if it remains immobile, it is a certain sign of death." Whatever may be the value of this sign, I should be very reluctant to see such an operation intrusted to unskilful hands.

The last division of ocular signs of death embraces those which are of certain value. The demonstration of the presence of the three images of Purkinje was first proposed by Legrand in 1850. In explanation of the method it will suffice to revert to what has been said about dimness of the cornea to comprehend that the disappearance of one, two, or even three of them is due to alterations that have been already described. It must be remembered, however, that often the eyes of the living do not permit of their demonstration.

Shall we be any more fortunate with ophthalmoscopy? The reader understands that we are concerned only with the signs observed in the first hours following death, before dimness and defects of the refractive media have made it impossible for luminous rays to penetrate. If at this very

moment the ophthalmoscopic mirror offers an indubitable sign of death, any restrictions of technique should not be complained of.

Who first applied ophthalmoscopy to the study of the signs of death is a question which, to the writer, remains obscure. The report of Devergie, already referred to, speaking of a candidate and another author, neither of whom he names, shows that in 1873 many observers had applied ophthalmoscopic study to the eyes of the dying. In 1868, Bouchut published observations made the year before, and presented them to the Academy of Sciences under the title, "A Memoir on some New Signs of Death furnished by the Ophthalmoscope." In 1878 he said that he had published in 1869 the observation which settled his conviction. This was corroborated by Meunier in 1868, and by Poncet, of Cluny, in 1869. To these and to a memoir of Gayat in the *Archives Générales de Médecine* for 1877 we are indebted for the best elucidation of the question.

In an atlas published in 1876, and entitled "Medical Ophthalmoscopy and Cerebroscopy," Bouchut introduced a picture (Pl. XIII., Fig. 3) marked "Signs of death found at the Fundus Oculi." In it can be seen opalescence of the retina and chorioid. In regard to it he says, "In this figure all of the fundus oculi is pearl gray, of an almost uniform tint, and the papilla is determined with difficulty. The disappearance of the retinal arteries and the interruption of the blood-current in the retinal veins are recognized by a pneumatosis similar to that which at times exists in the meningeal veins. There is discoloration of the papilla, which is gray like the rest of the fundus oculi. This appearance is constant in man and animals, evidencing the arrest of the circulation, and constituting one of the best and earliest signs of death."

In 1869, Poncet, of Strasburg, applied himself to the study of the ophthalmoscopic phenomena of death. For this purpose he devised a special instrument, of which a drawing is given in his memoir in the *Archives Générales de Médecine* for 1870. As the result of the examination of ten eyes in five subjects, made in the first hours after death, and facilitated by moistening the slightly dim cornea, he found that—(1) the retinal arteries usually disappeared, and the veins were generally represented by feeble, irregular, scarcely visible, broken streaks; (2) the chorioidal plexuses themselves disappeared, and were replaced by white or light red lines in the midst of a pigment which was more or less dark, according to the complexion of the subject; (3) the papilla was distinguished only with difficulty as a pale yellow disk, without other vascularity than some isolated or irregular streaks. From these observations Poncet endeavored to differentiate those that are found by post-mortem examination. He demonstrated that in syncope there were empty arteries and a paling of the veins. He states that the end of an attack in this class of cases is announced by the prompt return of the blood, the syncope producing a transient anæmia of the retina.

As to lethargy: a case of this type, in the service of Legrand, was

examined at the Bicêtre by Meyer and Bouchut. Here the vessels leaving the papilla were found to be of normal calibre. The vessels of the retina, without having changed in volume, were pale, and did not present their normal flexuosities.

Poncet also published many experiments on animals to show the disturbances that are produced in the fundus of the eye during the passage from life to death. As a result, he concluded,—

1. That in certain animals (oxen and sheep) the vessels are somewhat obscured by abundant dark pigment which invades the papilla, making the changes produced at the moment of death very hard to recognize.

2. That in others (dogs and rabbits) ophthalmoscopic examination reveals, as in man, sudden and characteristic changes.

3. That we must admit that in man there is in the appearance of the fundus of the eye a sign of death that is remarkable for its rapidity and certainty.

"This sign," he says, "is the general coloring of the fundus of the eye (which is red during life and yellowish at the instant of death) and the anæmic condition of the papilla." He found that the arteries disappear, and the veins are reduced to small, irregular, and filamentous lines.

With such valuable and precise evidence the question should be considered as settled. In 1876, however, Gayat detracted somewhat from his belief as to the certainty of the ophthalmoscopic signs. He informed us that in the course of a discussion held at the Congress of Heidelberg, Weber stated that in his experiments on animals, in which he had ligated the abdominal aorta, emptiness of all the intra-bulbar vessels occurred in four minutes, the retina being entirely exsanguinated. In these cases death shortly followed. He also found that similar manifestations were produced in animals of weak constitution in whom he had cut the splanchnic nerve, and who died soon after. In most of this series, however, the arteries in the periphery of the retina did not completely empty themselves.

A. Weber noted the division of the blood-column and the slowing of the blood-current, but he scarcely ever saw the arteries empty themselves. In other cases he found that death occurred without being preceded by any visible alteration in the retinal circulation. To this communication Becker added that, having examined many fresh eyes in the bodies of those who had recently died, he noticed that the arteries were full of blood.

These citations were confirmed by Gayat. He examined the eyes of five decapitated people, and found the above symptoms as long as the condition of the cornea permitted a view of the fundus of the eye. In four an orange-yellow tint of the fundus was recognized,—a condition which was not noted by Bouchut. Three times the arteries were almost empty, once empty on the papilla itself. The veins were full at the periphery, but their blood-columns were broken even in this situation. Gayat believed this to

be due to an infiltration which takes place along the vessels and in their sheaths. He also often noted a red spot in the macular region which he believed himself to have been the first to describe as one of the series of ocular changes found after death. This, he thought, should be classed with the phenomena that are observed in embolism of the central artery of the retina. In a decapitated white rabbit the retinal vessels were full of blood ten minutes after the fatal blow, and showed the same relations to one another in regard to color that they did during life. After half an hour the arteries were less full, and there was no alteration in the venous circulation. Examination of cadavers shortly after death gave no other results than those mentioned. In a phthisical patient seen one hour after death the fundus became yellow and the retinal vessels disappeared upon the disk. Examination during the period of coma demonstrated a considerable disproportion between the calibre of the arteries and that of the veins. A young man dead of typhoid fever, and kept in a temperature of ten degrees centigrade, showed retinal vessels that were full of blood two hours after death. Finally, a man of seventy-eight years, who was kept for six hours at a temperature of from six to ten degrees centigrade, showed retinal vessels that were normal.

In his memoir Gayat opposed Doe, whose opinion was that the interruption of the blood-current in the retina was a pathognomonic sign of death. Two years later, however, Gayat stated that in a patient seen by him this sign did not appear until twenty minutes after death. He attributed this fact to the patient's having been atheromatous and paraplegic.

What has been said of all the signs which we have studied is true of the interruption of the retinal circulation and the change of color of the fundus. Invested with positive value by those who first noticed the signs, they have lost their importance in proportion as they have been studied. Further, when the former condition of the patient's health, certain individual peculiarities, the mode of death, and, finally, the surroundings in which the body is kept, are investigated, the significance of any ocular signs varies.

It is pre-eminently the function of organic life—of the life of our tissues—to prevent the elements of which they are composed from undergoing the chemical changes which are characteristic of death; but life exists so differently in so many different and interrelated systems that it can never stop at the same time in all of them. It cannot be said that when a phenomenon of arrest in the vessels is noticed, the circulation has ceased and the ocular or vascular organ is dead, since every day resuscitations of such organs can be witnessed. It is only after a certain time, when the sign of each local death appears in turn and persists, that the conviction of general dissolution forces itself upon us. Public hygiene contents itself with demanding this conviction from all conscientious observers.

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